CAUSES OF BLINDNESS AND THEIR SOCIAL IMPLICATIONS

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Introduction

This book, Anatomy and Diseases of the Eye, is written specifically for the blind person who is a social worker for the blind, to give him concrete, working impressions of the eye, how it functions, and how it responds to different diseases. As part of "the case history," the social worker has an "eye examination." If one is able to understand "the eye examination" he will know much more about the person with whom he is working. He will know the course of the disease, what time of life it caused blindness, how rapid the onset of the blindness, the amount of the blindness, and just what chances there are of restoring some or all of the lost vision. This definite knowledge of the person's condition increases the social worker's interest in him and increases his confidence in the social worker.

In working up a case report, all sources of information are utilized. Some confusion may arise over "ophthalmologist, oculist, optometrist, and opticians." The words "ophthalmologist and oculist" have the same meaning. The term "ophthalmologist" although a bit long is replacing the term "oculist." An ophthalmologist first went through medical school, received his M. D. degree, and took an internship, just as your family physician did. He may have spent several years in general practice himself. Then he went on to specialize in diseases of the eye. For this he spent several more years in some medical center, studying this specialized field. On completing this special training he is qualified to examine eyes, to diagnose any diseased condition present, and to treat it in whatever way seems best, either medically or surgically. He also examines the eyes to find if glasses are necessary and if so just what glasses are needed.

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The optometrist is also a specially trained man. He has been to college and has received a degree of doctor of optometry, D. O. His training has had one purpose, to examine eyes to find whether or not they need glasses and if they do need glasses just what glasses are best for them, to give them the best vision. His training in diseases of the eyes is very limited and when anything abnormal is found he does not treat it. He sends these patients to an ophthalmologist for diagnosis and treatment.

The optician is the third man in the group. He makes the glasses. He makes them as directed by the ophthalmologist or the optometrist. He does not examine the eyes in any way.

These men are very frequently confused, but each has his separate field. The ophthalmologist is concerned with all phases of the eye, diagnosis and treatment, and examining of the eyes for glasses. The optometrist examines eyes for glasses only, and the optician makes the glasses as directed by the ophthalmologist or the optometrist.

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Anatomy and Physiology of the Eye

Anatomy is the study of the structures of the body, their size, shape, etc. Physiology is the study of what these structures do.

To learn the anatomy and physiology of the eye, we must not only consider the eyeball itself but the associated organs so necessary for its proper functioning. These include the orbit, the eyelids, the conjunctiva, the lacrimal apparatus, the muscles and fascias, the nerves, and the arteries.

The orbit is the bony cavity in the skull which holds and protects the eyeball and its associated structures. It is shaped like a four sided pyramid with the apex pointing back into the head. The base of the orbit, or front opening, in the average skull measures one and one-half inches in width and one and three-eighths inches in height. It averages one and one-half inches in depth. The inner side of the orbit runs straight back parallel to the inner wall on the other side. The outer wall extends backward and inward toward the inner wall, at a forty-five degree angle to it. The roof runs upward about an eighth of an inch and then backward and downward. The floor drops down and back for a short way and then goes upward and backward. When you examine a skull these walls feel very smooth. There are no sharp angles where they come together. Instead, they feel like a continuous curved surface. The walls are almost paper thin. Where they come together they are thicker and strong. Their outside edges at the base of the pyramid where they join the bones of the face become much thicker and very dense, forming a strong protective ring of bone around the opening into the orbit. This heavy ring of bone can easily be felt through the skin.

The bony orbit has several openings connecting it with the neighboring

spaces. Back at the apex of the orbit is an oval opening about half the size of a lead pencil leading into the cranial vault. This is the optic foramen. The optic nerve and the ophthalmic artery pass through here. Just in front of the optic foramen is a one-half inch long slit-like opening into the cranial vault between the roof and outside wall, called the superior orbital fissure. The nerves to the eye muscle enter through here. Below this a similar slit-like opening nearly an inch long, between the outside wall and the floor of the orbit, leads down behind the maxillary sinus. This is the inferior orbital fissure. The lacrimal canal is the other main opening into the orbit. It starts just behind the rim of the orbit where the floor and inner wall meet and runs down into the nose.

The walls of the orbit really act as partitions, giving equally important support to the neighboring structures. The roof of the orbit is also the floor of the anterior cranial fossa with the frontal lobe of the brain resting on it. Anteriorly it is often hollowed out by part of the frontal air sinus from the nose. The inner wall of the orbit also acts as the outer wall for the ethmoidal air sinuses. The floor of the orbit is the roof of the maxillary air sinus. And some of the jaw muscles are attached to the outside of the lateral wall.

The bones of the head grow together forming one solid complicated unit. For study and description, the anatomist takes them apart again, like a Chinese structural puzzle. Those that comprise the orbit include:

Roof - frontal and sphenoidal bones

Lateral wall - zygomatic, sphenoidal and frontal bones

Floor - maxillary, palatine, and zygomatic bones

Medial wall - frontal, lacrimal, maxillary, ethmoidal and sphenoidal bones.

The Eyelids

The eyelids are felt when you place your hand over the closed eye. They are about as thick as the edge of the fold between your thumb and index finger. Each eye has an upper and lower lid, the upper and lower lid having similar construction. Each lid has four layers. The outer layer, which you touch, is very thin loose skin. You can move it around, and pucker it up between your fingers. Having the skin very thin and loosely attached allows rapid motion of the lids. Because of this loose attachment the lids swell markedly when injured, as with a black eye. Under the skin lies a thin flat sheet of muscle, the orbicularis oculi, the fibers of which run in a circular fashion parallel to the edge of the lid. This muscle extends from the lid margins out beyond the bony rim of the orbit. It closes the eyelids by pulling in a puckering fashion, in the same way as the lips are closed. Underneath the muscle lies the tarsal plate. This tarsal plate is made up of dense fiber tissue which makes it as stiff as the cartilage in the nose. This stiff plate curves to fit snugly against the eyeball. At either end these tarsal plates have fibrous extensions. The upper and the lower fibrous extensions join together and attach to the bony wall. You can feel them past the outer end of the lid margins when the eye turns way toward the nose, and at the inner end of the lid margins when the eye turns outward. These fibrous extensions anchor the lids, and act like the hinges on a woman's purse. In the tarsal plate are the meibomian glands. They are little narrow tube-like glands which have their openings in a row along the lid margins, and run back parallel to one another in the tarsal plate. The last layer on the inside of the lid is the conjunctiva. It is a very thin, smooth covering, which is firmly attached to the tarsal plate so that it cannot wrinkle as the lid moves over the front of the eye.

Where the lids come together they have a flat surface, the lid margin. The front edge of this surface is curved and the lashes are located along it. There are about 100 to 150 lashes on each upper lid and 50 to 75 lashes on each lower lid. At the base of the lashes are sweat and sebaceous glands; the glands of Moll and Zeiss. The back edge of the lid margin, against the eyeball, is at a sharp right angle with the back surface of the lid, giving a firm smooth closure of the eyelids over the eyeball.

The lids stand constant guard over the eyeball. They close completely by reflex control at signs of gross danger as a flying object, a very bright flash of light, a loud noise, falling, etc., or any touch to the eye itself, the eyelashes, or the skin of the lids or the face nearby. They close part way, i.e. squinting, to protect the eyes from dust. This way the lashes stop the particles but vision is possible. In bright light the lids close part way, cutting down the amount of light entering the eye as too much light hurts or even damages the eye. Also some persons with high errors of refraction can see better by squinting.

By winking, rapidly closing and opening, the lid acts like the windshield wiper on a car, cleaning dust, mucous, etc., off the front of the eyeball. Also winking carries the tears across the front of the eye. These tears help nourish the front of the eyeball, the cornea, and by keeping it wet and smooth maintain the best optical surface for vision.

When asleep the lids close over the eye protecting it and keeping it moist.

The lids also give the eyes most of their expression.

Conjunctiva

The back of the eyelids and the front of the eye are covered by a thin

mucous membrane. This is very similar to the membrane on the inside of the mouth. By feeling the inside of the lip one can see how smooth and slippery it is, especially against the moist tongue, which is similar to the lid moving over the eye. The conjunctiva starts at the lid margin and covers the back of the lids. As it goes over the tarsal plates it is bound as closely to them as the cloth is to the hard cover of a book. Beyond the tarsal plates it has several loose folds. These loose folds allow the lid to move freely without binding it at all. Here it turns back, coming forward over the eyeball. The potential spaces formed by this folding back on itself are called the superior and inferior fornices, or cul-de-sacs. As it comes on forward again over the eyeball, it is loosely attached. This loose attachment to the eyeball allows the eye to move freely, just as the lids were allowed to move freely by the folds above. This attachment becomes increasingly firm as it goes forward until the conjunctiva ends at the cornea or window of the eye. Here the conjunctiva is again bound down firmly. By being bound down firmly here it is prevented from folding forward, over the front of the eye. The conjunctiva is often spoken of as the conjunctival sac. It can be thought of like a small rubber balloon. If you held the balloon in your open left hand, then pressed in on the other side with your right fist until your hands met, and then let the air out of the balloon, you would have a similar arrangement; with your left hand taking the part of the lids, your right hand the part of the eyeball, and the loose folded edges of the collapsed balloon representing the fornices, or cul de sacs. In the fornices the conjunctiva have several small glands which are similar to the lacrimal, or tear, glands. Then, all through the conjunctiva are special mucous cells which secrete a sticky material. This mixes with the tears to lubricate the conjunctiva and the cornea. At the inner corner is a small mound of tissue called the caruncle. This sometimes

has a few hairs growing from it. Just outside of it is a small vertical fold of conjunctival-like tissue called the plica semilunaris. This plica semilunaris is a vestigal remnant of the third eyelid as is seen in some reptiles and birds, where it acts as an extra protection.

The Lacrimal System

The lacrimal system is the body's arrangement for making, using and disposing of its tears. The tears are secreted by the lacrimal gland. This is located above the outer canthus, resting up against the roof of the orbit in a little depression just behind the orbital rim. It has a main portion and an extra, accessory gland. The main gland is almond shaped with its upper flat side facing upward and forward against the roof of the orbit and its lower edge pointing down toward the upper fornix of the conjunctival sac. The accessory gland lies behind and below the external palpebral ligament. The main gland has ten or twelve small ducts which pass down along side the accessory gland, and on to the edge of the fornix of the conjunctival sac.

The lacrimal gland will secrete about sixteen drops a day. From birth on the flow is increased by any irritation to the eye, acting through reflex stimulation. From about the third month tears are also formed as an expression of emotion as in crying, or sadness.

The tears flow down over the front of the eye and toward the inner canthus partly by gravity but mainly by the action of the lids in winking. The upper lid spreads them evenly over the front of the eye just as you spread soap over the back of one hand with the palm of the other. The oily secretion from the meibomian glands floats in a very thin layer over the layer of tears. In this way it prevents the tears themselves from evaporating. This oily secretion from the meibomian gland also covers the lid margins preventing the tears from flowing out over the lids and onto the cheeks.

Tears have several functions. As they pass over the eye they float along small particles of dust. When there is more material than the normal flow can wash away the accumulation of debris irritates the cornea. This irritation causes a reflex stimulation of the gland bringing a flood of tears to wash out the material. The tears carry nourishment to the cornea which has no blood supply. They form a very smooth surface over the front of the cornea making it a more perfect optical instrument. And they contain enzymes which inhibit the growth of bacteria. Also, they act as a potent form of expression.

Some of the tears flow along the inner edge of the lower lid in the angle it forms with the front of the eyeball, the rest go down to the inferior fornix and pass along it toward the inner canthus or angle of the eye. Because foreign material is so often found being washed along the inferior fornix, it is sometimes called the gutter of the eye.

At the inner canthus the lids stand a little way away from the eyeball, leaving a space in which the tears gather. This is called the lacrimal lake. From here they leave the eye to pass on down into the nose through the lacrimal passage.

This passage has three parts: the canaliculi, the lacrimal sac, and the nasal lacrimal duct. The canaliculi are tiny tubes, one in each lid which starts on the inner edge of the lid margin less than an eighth of an inch from the inner end of the lid. This opening is called the punctum. It juts out slightly into the lacrimal lake where it collects the tears. It is surrounded by a small ring of muscle which relaxes allowing the punctum to open, and the tears to enter, then tightens like a purse string to prevent them from getting back out. The canaliculi then run along the lid margin just under the skin to the inner angle of the eye. From here they go backward and inward behind the internal canthal ligament and enter the lacrimal sac.

The lacrimal sac lies behind the internal palpebral ligament. Its top extends just above the ligament, and it goes downward to the bony canal, the nasal lacrimal duct, leading into the nose. The lacrimal sac is about one-third of an inch long and half the diameter of a lead pencil. Muscle fibers of the orbicularis muscle separate to go both behind, and in front of, the sac, thus making a flat sheet of muscle on each side of the sac. When the muscle is relaxed, the sac is flattened from before backward. When the orbicularis tightens up, the walls are pulled taut, thus separating them and making a suction in the sac, which pulls the tears into the canaliculi. When the orbicularis relaxes, the muscles come together again and the tears run on down into the nose through the nasal lacrimal duct. This duct is a direct continuation of the sac down through the bone for about one-half inch to open through the lateral wall of the nose, near the floor of the nose. Its lining is a direct continuation of the lining of the lacrimal sac.

The Extrinsic Eye Muscles

The extrinsic eye muscles are the muscles located outside of the eyeball, with the intrinsic muscles being those located inside the eyeball. The extrinsic eye muscles are the ones which move the eye when a person looks in different directions. Although they are flat, ribbon-like structures, about one-half inch wide and about 1/60 of an inch thick, for their size they are both the strongest muscles in the body, and have the largest proportion of nerve fibers.

There are six of these muscles for each eye. Four of them start from a ring of fibrous tissue around the optic foramen at the apex of the orbit. These are the medial rectus, the inferior rectus, the lateral rectus, and the superior rectus. From this fibrous ring they diverge, running forward parallel

to their respective orbital walls to the equator of the eyeball, then they lay against the eyeball as they come on forward to end at their attachments into the eyeball, about a quarter of an inch from the cornea. These diverging muscles have somewhat the shape of a cone with its apex at the fibrous circle, and are often spoken of as the muscle cone. They can well be compared to an ice cream cone, the muscle cone being the wafer of the cone, and the round eyeball being the full round dipper of ice cream. There are also two oblique muscles which help move the eyeball, the superior oblique, and the inferior oblique. The superior oblique starts from the back of the orbit just above and medial to the fibrous ring and runs forward, medialward and upward to just behind the bony rim of the orbit at the inner end of the upper portion. Here it ends in a round, smooth tendon, which passes through a small fibrous pulley, to turn back and down. It runs back and outward, under the superior rectus muscle to attach to the eyeball. The pulley can often be felt through the skin of the upper lid as a small hard bump, and the tendon can be felt to tighten when the eyes turn down and in. The inferior oblique starts just inside of the bony orbit below, and opposite the pulley of the superior oblique. It runs backward and outward, under the inferior rectus to attach to the eyeball.

One other muscle in this group, the levator palpebrum acts on the upper eyelid rather than the eyeball itself. It starts above the ring of fibrous tissue, then runs forward as a flat ribbon just above the superior rectus muscle. Anteriorally, it splits horizontally into an upper and lower portion. The lower layer fans out and attaches to the upper edge of the upper lid. It acts right with the superior rectus, raising the upper lid when the eye is turned up. Fibers from its under side insert into the upper fornix pulling the conjunctiva up out of the way when the eye is turned up. The

upper layer goes forward ending in the skin of the upper lid. This portion pulls the skin up in a deep fold when the lid is raised. Otherwise this loose skin would fall forward as the tarsal plate was pulled up under it.

The extraocular muscles have a very exact control over the eyeballs. In turning, its center of rotation is always very near its anatomical center. Its motions are thought of as being (1) vertical, up and down, (2) horizontal, to the left and right, and (3) torsional, or a wheel rotation, clockwise or counter-clockwise. Actually nearly all motions are a combination of these three, and a study of all six muscles in their many positions becomes very complex. However, the main action of each muscle is easy to understand from the position of the muscle. The superior rectus turns the eye up, the medial rectus turns the eye in, the inferior rectus turns the eye down, the lateral rectus turns the eye out. The superior oblique helps to turn the eye out and down, and also has a torsional effect pulling the upper part of the eye in, giving a clockwise rotation to the right eye, and a counter-clockwise rotation to the left eye. The inferior oblique has the opposite pull, helping to turn the eye up and out and giving a counter-clockwise rotation to the right eye and a clockwise rotation to the left eye.

The nerve centers controlling the position of the two eyes normally keep them pointed in the same direction at all times. They always move together like a couple waltzing. One eye cannot look up and the other down, nor one in and the other out. The only exception comes on looking at a close object. Here the eyes turn in toward this object, convergence, but they are still working together as a team, both eyes looking at the same object.

The Fascias

All of the organs of the body are covered and bound together by one kind of tissue, appropriately called "connective tissue." Like the modern plastics it takes on many forms. In some places it is strong and dense, in others it is loose and fragile. And, depending on its use, it has been given many names. Where it holds the bones together at the joints it is called a ligament. Where it holds the muscle to its insertion it is called a tendon. Where it covers a muscle it is called the muscle sheath. Where it covers bone it is called the periosteum. The eye and its accessory structures are no exception. Its bony orbit is lined by a sheath of periosteum. This periosteum, or sheath of connective tissue, can be peeled off readily except at the line of junctions of the different bones. At the openings into the orbit, the periosteum continues out through them over the bone. The periosteum forms a sheath across each orbital fissure. Around the optic foramen it forms a dense ring, the Annulus of Zin, to which the four recti muscles are attached. The tendons of these muscles are a direct continuation of the connective tissue in this ring. Also, from this ring a layer of the same tissue goes forward over each muscle. The optic nerve also has a similar sheath. The eyeball itself from just behind the cornea on back is covered by another layer of connective tissue. This layer surrounding the eyeball is called Tenon's Capsule. When the muscle reaches the eyeball its tendon fuses into the outer wall of the eyeball and the muscle sheaths become part of Tenon's Capsule. Thus Tenon's Capsule becomes something like a glove, the hand portion being the Capsule proper, covering the eyeball, and the fingers being the sheaths of the muscle which extend directly from the Tenon's Capsule out over each muscle.

The Vessels

The circulation of the blood in the body has three parts. The arterial system carrying blood from the heart to the organs of the body, the venous system carrying blood from the organs back to the heart, and the network of fine capillaries between. The arteries constantly divide into smaller and smaller ones, like a river breaking up as it flows into a swamp, until the finest divide into a very fine network. This network is called the capillaries. Then these capillaries start joining together forming bigger and bigger vessels, the veins. Just as water flowing out of a swamp has small streams which join to make larger streams and then these join to make still a larger stream. In the capillaries the oxygen and nutrient substances leave the blood, while carbon dioxide, and other waste products are picked up. The blood in the arteries is under pressure from the pumping of the heart, whereas the returning blood in the veins has and needs very little pressure as it flows into the bigger vessels back to the heart.

The artery to the head, the common carotid, can be felt pulsating deep on the side of the neck, one on each side. Under the angle of the jaw it divides into the external carotid which supplies mostly the organs outside the skull, and the internal carotid which goes on up into the skull to the brain and structures inside the skull.

The ophthalmic artery comes off the internal carotid as this artery passes the optic nerve inside the cranial vault. The ophthalmic artery runs along under the optic nerve through the optic canal entering the orbital cavity inside the muscle cone. Here it swings out lateralward, then up and over the optic nerve. From here it runs forward and medially (medially means

toward the midline of the body) to the inner angle of the eye where it ends by dividing into a frontal branch going up to the forehead and a nasal branch going down to the nose, and two palpebral branches, one to each lid. As it goes along it gives off several branches. The central retinal artery comes off just inside the orbit, and runs along the optic nerve to about one-half inch from the eyeball where it enters the optic nerve and runs on forward into the eye. Next the posterior ciliary arteries, ten to twenty in number, come off and run along the optic nerve to the eyeball. Two of them go clear around forward to the ciliary body, which will be described later. The rest go directly into the choroidal circulation, which also will be described later. The lacrimal artery starts along side the optic nerve and goes forward along the upper edge of the lateral rectus muscle to supply the lacrimal gland. It sends a palpebral branch to the upper lid and one to the lower lid. Two main muscle branches are next given off, a medial and a lateral. These divide to supply the extra ocular muscles. There are other minor branches and all of the arteries give off small branches to the tissues around them, as they pass through. The veins are quite variable, but generally the follow the same pattern as the arteries with corresponding branches coming back to form the superior ophthalmic vein which leaves the orbit through the superior orbital fissure. The veins from the lower portion of the orbit form the inferior ophthalmic vein, which either joins the superior ophthalmic vein, or leaves separately through the inferior orbital fissure. One vein often has a branch running to another vein making a direct continuation. Thus the veins of the nose, which go down over the face may have branches running to the superior orbital vein. This makes a direct path for infection from the nose back through the orbit to the cranial vault. Also, when a vein is blocked the blood can flow out through another vein, like a detour on a highway.

The Nervous System

The nervous system of the body has two great divisions, the voluntary system and the involuntary system. The voluntary nerve system is that which is conducted at a conscious or higher level and includes the pathways by which sensations come into the nervous system and the pathways by which impulses are sent out to the muscles of the body. The involuntary nervous system, or autonomic nervous system, functions below the conscious level. It also has sensory routes, bringing sensations in, and motor routes which carry impulses out. This system functions without any conscious effort on our part, as is demonstrated by the beating of the heart, the actions of the stomach and intestines, etc.

The Motor Nerves

There are two types of nerves, motor and sensory. The motor nerves carry impulses to an organ from the nerve centers, while sensory nerves carry sensations from the organs to the nerve centers. The motor nerves of the eye are called the third cranial nerve, or oculo-motor nerve, the fourth cranial nerve, or the trochlear, and the sixth cranial nerve, or the abducens. They all arise from nuclei low in the brain. These nuclei are grouped in a line along the midline of the brain, so that anything which damages one may easily reach across to one on the other side. After leaving the brain substance, the nerves run forward along the base of the brain to the superior orbital fissure. The sixth cranial nerve has the longest way to go, so is most apt to be involved in injuries or diseases in this area.

The third cranial nerve passes through the inner end of the superior orbital fissure, and enters the orbit through the common ring tendon, or Annulus of Zin. Here it divides into two parts. The superior division goes up to the superior rectus muscle then sends a branch on through this muscle to the levator palpebral muscle. The inferior division sends branches to the medial rectus muscle, the inferior rectus muscle, and the inferior oblique muscle.

The fourth cranial nerve, or trochlear nerve, comes through the superior orbital fissure outside the common ring tendon and runs forward and inward to supply the superior oblique muscle. Trochlea means pulley. The nerve was named after the pulley of the superior oblique muscle.

The sixth cranial nerve, or abducens nerve, comes through the inner end of the superior orbital fissure and the common ring tendon, and passes directly to the external rectus muscle. It is called abducens to describe the action of the external muscle. It abducts, moves out, the eye.

The Sensory Nerves

The sensory nerves of the eye and the surrounding structures are supplied by the fifth cranial nerve, or tri-facial nerve. This nerve has three branches, the ophthalmic division, the maxillary division and the mandibular division which supply approximately the upper, middle, and lower portions of the face in their respective orders named. The eye and its structures receive branches from the first two divisions. They all start in a "ganglion" called the semi-lunar or gasserian ganglion. A ganglion is a collection of nerve cells which act as a relay station in the nerve pathway. The first portion, or ophthalmic division, starts in the gasserian ganglion and passes forward to the superior orbital fissure where it enters the orbit and divides

into three main branches, the lacrimal, the frontal and the nasal ciliary branches. The lacrimal nerve passes along the lateral wall of the orbit to the lacrimal gland and on out just above the lateral canthal ligament to supply the skin of the face in that area. The frontal nerve goes forward along the roof of the orbit to the supra-orbital notch, a small notch in the orbital rim and then passes upward on to the forehead to supply the skin in this area. The nasal ciliary branch enters the orbit through the common ring tendon into the muscle cone, just lateral to the optic nerve. It passes over the optic nerve, then the medial rectus muscle. From here it has a peculiar tortuous route. It goes through the medial wall of the orbit to a small opening into the ethmoid cells, then up to the cranial vault where it passes for a short way along the floor of the cranial vault, then turns back down to enter into the nose where it divides into nasal branches. Near the optic nerve, the nasal ciliary nerve gives off branches which go forward to the eyeball. Just before it enters the ethmoidal foramen the nasal ciliary nerve gives off a branch, the infratrochlear nerve, which goes forward to supply the skin of the upper eyelid, the nose and the lacrimal sac. The second division of the cranial nerve, the maxillary nerve, enters the orbit through the inferior orbital fissure and passes along the floor of the orbit in a groove and about half way forward this groove passes into a canal which runs just under the floor of the orbit forward and out onto the cheek through an opening called the infra-orbital foramen. Here it sends branches to the lower lid, the lacrimal sac, the sides of the nose, the upper lids, and the cheek.

The Autonomic Nervous System

The autonomic nervous system, as was explained before, controls those body functions which are not consciously controlled. In the eye it controls

the rate of flow of blood into and out of the eye by controlling the size of the arterioles, the capillaries and the venules. It controls the rate of flow of the aqueous into the eye, and it controls the size of the pupil and the state of contraction of the ciliary muscle. Also, by controlling the above factors, it controls the state of tension inside the eye.

The autonomic nervous system has two divisions, the sympathetic division and the parasympathetic division which always act in an opposite fashion. The sympathetic nerves arise way down in the spinal cord, pass upward in front of the vertebral column along what is known as the sympathetic trunk, through several ganglia in the neck to the carotid artery where they form a dense network around it known as the carotid plexes. From here they travel by a complex route along the arteries going up near the ear and then forward again. Some reach the eye with branches of the ophthalmic nerve through the nasal ciliary nerve, others go to the ciliary ganglion and then forward with its nerves, the short ciliary nerves, to the eyeball. These act to dilate the pupil of the eye. The parasympathetic nerves arise in the brain near the nucleus of the third nerve and their fibers pass forward with the ocular motor nerve, to reach the ciliary ganglion. A ganglion is a collection of nerve cells which act as a relay station for the nerve impulses. From the ciliary ganglion fibers run forward as the short ciliary nerve to the eyeball where they enervate the ciliary muscle and the sphincture muscle of the pupil to make them small.

The Eyeball

The eyeball, the organ of vision proper, is a sphere, two thirds the size of a ping pong ball, set in the front part of the orbit. It is almost perfectly round. The front portion, the cornea, has a sharper curve so it

juts out a bit much like a blister would on the pad of your finger. In size the eyeball measures just an inch in diameter. In volume it fills about one-fifth of the bony orbit. It sits in the front part of the bony orbit, so that a pencil held vertically against the front of the orbit, touching the upper and lower margins, generally presses slightly against the eye through the closed lids. It sits slightly up and out from center.

The outer wall of the eyeball is a very dense fibrous coat with two separate portions. The front portion, or cornea, is transparent. The posterior five-sixths, the sclera, is white and opaque. Inside the eyeball against the sclera is a layer of blood vessels, the vascular coat, and inside this lies the retina. These three layers lie one on another like the layers of an onion. Inside the eyeball behind the cornea is a space called the anterior chamber. Behind this space lies the iris and the lens. These fill the front fifth of the eyeball. The remaining four-fifths is filled by a jelly like substance, called the vitreous.

The Cornea

The fibrous coat of the eye is about as thick as the cover on a leather notebook, and about as tough. The name Cornea comes from the Greek word meaning horny, and the name Sclera comes from the Greek word meaning hard.

The cornea is the window of the eye. Light passes freely through it without any distortion. This way it acts like the crystal on a watch, letting light in yet giving strong protection to the eye. As the cornea has a sharper curve than the sclera, where the two meet they form a groove, the scleral sulcus. The circle of tissue where the two join is called the limbus.

The cornea has five layers. The first or outside layer is the epithedial layer. This is a direct continuation of the conjunctival epitheleum as it

comes over the eyeball, and is a modified form of the same epitheleum that covers the rest of the body. Here it thins out into a smooth layer, five cells deep. It lays very flat against, and firmly attached to the next layer, or Bowman's Membrane. Under a microscope, this membrane appears as a thin structureless sheet. The next layer, called the substantia propia, makes up nine-tenths of the cornea. It is composed of parallel fibers of connective tissue. This arrangement of the fibers prevents disturbance of the light as it passes through. Against the substantia propia on the inside lies the fourth layer, another thin structureless membrane, Decemet's Membrane. The fifth layer, and the inside layer, is a single layer of flat cells, called the endothelial layer.

The cornea itself has no blood vessels. So it must get its nourishment in a more indirect fashion. The subconjunctival tissues, in the area around the cornea, have a rich blood supply, so some serum gets to the cornea from there. Some nourishment comes from the tears which are passing over it and some nourishment is obtained from the aqueous behind it.

The surface of the cornea has more nerve endings per square millimeter than any other surface of the body. These are mainly fibers of pain, with some warm and cold fibers. This large number of pain fibers is to give the best possible protection to the cornea.

The Sclera

The sclera forms all of the rest of the dense fibrous coat of the eye. It is also formed by connective tissue. These long connective tissue fibers are closely interlaced as they are laid down in all directions running around the eye. This tissue has very few blood vessels. The tendons of the muscles as they are inserted into the sclera have their fibers blending right into the

fibers of the sclera itself. The scleral coat has few openings. There are four openings around the equator, the equator being the diameter running around the top, sides, and bottom, for the veins of the choroid, called the vena vorticosa. At the posterior pole of the eye the sclera has a large opening for the optic nerve and numerous small openings for the ciliary nerves, and arteries. At the opening for the optic nerve the fibers do not part and leave one large opening, rather they separate into small bundles which cross in an interlacing fashion very similar to the pattern of a window screen. In this way the fibers of the optic nerve can pass through the openings and still the meshwork of sclera fibers gives support to the eyeball in this area. However, this scleral meshwork, called the lamina cribosa, is weaker than the rest of the scleral coat, and, as will be described later, when the pressure inside of the eye becomes greater, this is the first place to give way.

Vascular Tunic

This vascular tunic is the second coat of the eye. It is made up of three portions, the choroid, the ciliary body, and the iris. The choroid lays flat against the sclera over the posterior two-thirds of the eye. It is composed of blood vessels. The outer portion has larger vessels and towards the inside are interwoven capillaries. Their function is to give nourishment to the outer portion of the retina, the innermost layer of the three coats. The choroid is very loosely attached to the sclera, so that in certain diseased conditions it readily separates from the sclera. On the other hand its inner surface is very closely adherent to the retina.

As the choroidal layer comes forward it blends into the ciliary body. The network of vessels is gradually replaced by thin strands running forward. This layer gradually becomes thicker so that on cross section it has the appear-

ance of a triangle, one side being against the sclera, the second side facing inward, and the third side facing forward toward the anterior chamber. As it thickens, the ciliary body becomes composed of smooth muscle fibers. Smooth muscle fibers are controlled by the autonomic or involuntary nervous system. Under a microscope they are found to be long, tapering off to a point at either end. Some of the muscles run parallel to the sclera. Some radiate inward from the anterior edge of the ciliary body next to the sclera. But the vast majority go around the eye in a circular fashion. For this reason the ciliary body is often thought of as a ring of muscle and for practical purposes it is just that, a ring like a wedding band, of uniform thickness going around the eye against the anterior portion of the sclera. The junction of the choroid and the ciliary body is known as the ora serrata. The first portion of the ciliary body as it goes forward is thin and flat, and is known as the pars planum. The anterior two-thirds of the ciliary body, where it becomes thicker, is covered on its inner surface by deep radial folds called the corona ciliaris. The fluid of the eye, the aqueous, comes from these ciliary processes. Whether the aqueous is actually secreted or is formed by some physical process has not been determined, but it is easiest to think of it as being secreted here. Arising in the folds of valleys between these processes are many very fine delicate strands, the suspensory ligaments. These run forward toward the center of the eye to the lens where they are attached, under tension, to the capsule of the lens, and act like guy wires. The proportions are poor, but these can be thought of as being similar to the spokes on a bicycle wheel running to the hub at the center and holding the hub in position. When this ring of muscle in the ciliary body contracts down it makes the ring smaller and thus loosens the tension on these guy wires, the suspensory ligaments. This in turn loosens the tension on the capsule of the lens, allowing the lens which is quite elastic to become more spherical.

The iris is the third portion of this vascular tunic. The iris is a flat, disc-like membrane with a hole in its center, the pupil. The outer edge of the iris is attached to the ciliary body. On its posterior surface it is covered by a thin layer of densely pigmented cells, the pigment epithelium, which is a direct extension of the inner coat of the eye, the retina. On the pigment epithelium are radial fibers of smooth muscle. Over this is the iris stroma, a very delicate layer of blood vessels and loose connective tissue forming the main portion of the iris. At the inner edge of the iris is another circular muscle which runs around the edge of the pupil in the back of the iris against the pigment epithelium layer. The iris lies against the lens so that the central portion is a little more forward than the outer portion giving it somewhat the shape of a saucer with a hole in the center. The ring muscle, the sphincter muscle of the iris, which lies around the edge of the pupil makes the pupil small as it contracts down. On the other hand the radial muscle fibers, the dilator muscle, as its fibers contract, pull the pupil wider open. The iris stroma itself is so delicate that it offers no resistance to the pull of these muscles. The iris thus acts like a curtain for the eye, or like the diaphragm on a camera, opening readily to let in more light or constricting down rapidly to cut down on the amount of light getting into the eye, or to give a sharper focus optically to the eye. Although the pigment epithelium is just two cells thick, these cells are so densely packed with pigment they prevent the passage of light and thus allow the iris to act effectively as a curtain.

The Anterior Chamber

The anterior chamber is the space between the cornea and the iris. It can be thought of as a plastic cup placed with the cup side against the back

of a saucer, the center of the saucer being the pupil of the eye, the saucer itself the iris, and the plastic cup the transparent cornea. The space under the cup, between the cup and the saucer, would be the anterior chamber. The anterior chamber contains clear fluid known as the aqueous. This aqueous is formed in the ciliary processes. It comes forward between the iris and the lens, through the pupil into the anterior chamber and from here it goes to what is known as the angle of the anterior chamber at the junction of the cornea and the iris where it passes out into the blood circulation again. This aqueous is perfectly transparent, allowing light to pass through it freely. It carries nourishment to the posterior portion of the cornea, and it carries nourishment to the lens.

The Chamber Angle

This is a very important area in the eye. It is formed where the cornea and the iris are joined. Reviewing briefly the anatomy of the eye, most of the outer coat is made up by the sclera. The anterior sixth is composed of the cornea which has a sharper curve so that it bulges forward somewhat on the front of the eye. Inside, against the sclera, is the vascular coat. The anterior portion of this is the ciliary body, extending up to the junction of the cornea and the sclera. Then, coming in from this, hanging like a diaphragm across the front of the eye with a slight bulge forward and the pupil in its center is the iris. On examining a cross section of the eye the anterior chamber is seen to end in a sharp angle at each side, with the cornea as the front side of the angle and the iris and a bit of the ciliary body as the back side of the angle. This is called the chamber angle. It is the outer edge of the anterior chamber. All around the apex of this angle is a fine meshwork of fibrils called the pectinate ligaments. The spaces between

them are called the spaces of Fontana. Just outside these spaces in the junction between the cornea and the sclera is a very fine canal which runs all around the front of the eye in the corneal scleral junction. This is called Schlemm's canal. From Schlemm's canal there are direct continuations into the ciliary veins. This is the normal outflow of the aqueous. However, it can be blocked in many ways. If the iris is pushed forward against the cornea, the aqueous cannot pass on into the spaces between the pectinate ligaments and out through Schlemm's canal into the ciliary veins. The spaces between the pectinate ligaments are very small and can be blocked readily by inflammatory material, that is the cells of inflammation which will be discussed later, or fibron, etc. Also they can be blocked by blood from a hemorrhage, by tumorous cells or any other material. Any form of blockage here at the angle prevents the aqueous from leaving the eye. However, the aqueous continues to form causing an increase in intraocular tension above normal. For that reason this angle area is very important to the health of the eye.

The Retinal Coat

The innermost of the three layers is called the retina. This retinal tissue lines the whole inside of the eye, starting with the border of the pupil and extending all the way back to the optic nerve. Just as the vascular coat lies directly against the sclera, so the retina falls directly against the vascular coat. The portion on the back of the iris has already been described as being two cell layers thick, the cells being densely packed with pigment. This layer extends directly on back over the ciliary body. Here, again, it is composed of two layers of cells. The layer directly against the ciliary body is, like those in back of the iris, densely pigmented. The inside layer of cells does not contain pigment. The two layers are adherent to one another.

On back, at the ora serrata, the true retina begins. Here there are two main divisions, the outer pigmented layer and the retina proper. The outer pigmented layer is called the pigment epitheleum of the retina and consists of a single layer of cells containing a considerable amount of pigment. The pigment epitheleum is bound tightly to the choroid by a clear structureless membrane called the membrane of Bruk, but is very loosely attached to the retina proper.

The retina proper is the sensory layer of the eye. It is this layer which receives the impressions, or stimulation, of light and transforms them into nervous energy, this nervous energy being in turn transferred on up into the brain where it is interpreted. The retina is the king structure of the eye. All of the other structures of the eye and of the orbit function to hold this layer in place, protect it, carry nourishment to it, and to focus the light upon it properly.

Grossly it lays very flat and smooth against the inside of the eye. It is readily separated from the pigment epitheleum and when separated is found to be a most delicate thin membrane, much thinner and more delicate than the membrane you find just under the shell when you peel a hardboiled egg. It has a uniform appearance. At the optic nerve the central ophthalmic artery immediately divides into a superior and inferior branch, each of which in turn before leaving the disc, or optic nerve head, divides again into a nasal and temporal branch. The veins are similarly arranged. At the back of the eye, just temporal and a little below the entrance of the optic nerve is a small depression known as the fovea. This is the point of central vision where the visual acuity is the highest.

As stated previously the retina is a direct outgrowth from the brain substance. Functionally the retina contains three layers of nerves. A nerve

receives a nervous impulse or message and then sends it on. It has three parts, the cell body proper and two processes, one to receive the impulse and the other for sending the impulse on forward. These processes can be very short or sometimes are very long, and groups of them together form what we generally think of as nerves.

Here in the retina there is the outer layer of nerve cells next to the rods and cones where the impulse of vision originates. This passes up to a second layer of cells and from there it is passed on inward to a third layer of cells. These cells, of course, have short processes, but the inner layer has a long process for sending the impulse on up to the brain. Light coming into the eye passes through the entire thickness of the retina to the outer layer of the retina proper. This layer, known as the layer of rods and cones converts the light into a nervous impulse, sending it up the pathway just described.

Studied under the microscope the retina is seen to have ten different layers. It is easiest to describe these as starting from the outside in. The first layer, the one against the choroid, or vascular coat has already been described as the pigment epitheleum. Just inside the pigment epitheleum is the layer of rods and cones. This is the sensory layer of the retina where the stimulation is received by the sensory endings, the rods and cones. The cones as their name implies are rather coned-shaped. The rods, on the other hand, are just as their name implies, rod shaped processes. These lay side by side extending from the pigment epitheleum in to a clear thin membrane called the external limiting membrane. Inside of this external limiting membrane are the nuclei of the rods and cones. The nuclei as a group are called the outer nuclear layer, and under the microscope appear as a layer of round dots about five or six nuclei deep. The nerve fibers going in from this outer nuclear layer form a meshwork in appearance called the outer molecular layer

or outer plexiform layer. These nerve fibers go into the nuclei of the second group of nerves. These form another layer called the inner nuclear layer which is about five nuclei thick. Their neurons in turn pass on inward forming the inner molecular layer. They go to the third layer of nerves called the ganglion cell layer. The ganglion cells have larger nuclei being only one or two nuclei thick so that they receive impulses from several nerves of the inner nuclei layer. The nerve fibers from the ganglion cells are very long and pass along the inner surface of the retina toward the optic nerve, forming the stratum opticum or nerve fiber layer. Then, inside this nerve fiber layer is a thin structureless membrane called the internal limiting membrane between retina and the vitreous.

The retina is of uniform character throughout except at the edges of the ora serrata and at the macula or point of highest vision. In the macular area, the layer of rods and cones loses most of its rods. The rods are used mainly for night vision and to detect motion but not for sharp vision. The cones increase in number to replace the rods. The cones are used for finer, more distinct vision and entirely for daytime vision. The very center known as the fovea centralis has no rods, and the other layers of the retina thin out leaving only a few nuclei of the outer nuclear layer. By eliminating the other layers here and having the nerve fibers stretch out to them along the edge of the macula there is less interference with light giving a much sharper optical image to focus upon the cones. Near the ora serrata, or anterior edge of the retina the retina begins to thin and to have fewer rods, cones and nerves and more supporting fibers. This area is normally blind or with very low visual acuity. From the age of 35 on it often develops cysts. These cysts are sometimes thought to be the cause of retinal detachments, as they rupture and allow the fluid inside the eye to get in behind the retina and force it away from the pigment layer.

The Lens

The lens is situated behind the iris where it is supported by the zonular fibers as previously described. It is about the size of a small lima bean. Seen from the front it is round. Seen from the side the front surface has a slight curve and the back surface a moderate curve. When you cup your left hand moderately and your right hand slightly and place them together against each other, you have about the same curve, the left hand being the back curve and the right hand covering the front surface. The lens is quite elastic, so, when the ciliary muscle contracts allowing the zonular fibers to relax, the lens in turn loses their stretching effect and become more curved. On the other hand when the circular portion of this ciliary muscle relaxes, these fibers are made more taut and put more of a tension on the lens capsule and thus flatten the curve of the lens. It is by this process that the lens focuses rays of light upon the retina. Under a microscope the zonules can be seen coming up to the lens where they insert into the lens capsule. This capsule has a perfectly clear structureless appearance. Under the capsule, over the anterior half of the lens is a single layer of cells, called the lens epitheleum. This extends out to the edge or equator of the lens. Instead of extending on around back of the lens these cells from here are scattered in through the substance of the middle of the lens. The substance of a lens proper is made up of many layers of fibers. These fibers are laid down parallel to one another forming layers much like the layers inside of an onion, one upon the other.

Vitreous

The vitreous fills the remaining four-fifths of the inside of the eyeball. On examination, this vitreous is perfectly colorless and clear, and is about the same consistency as medium thick jelly, or thin jello. It is perfectly structureless when examined under a microscope, except for a few strands which are formed by the fixation fluid used in making the slides. Studied chemically it has almost the same composition as the aqueous except for a small amount of material called muco protein which makes it jell, much like a small amount of gelatine added to water makes a large body of semi-solid gelatine. The vitreous is a little more condensed in its structure at its surfaces forming a sort of a membrane. This is not attached anywhere except at the nerve head or entrance of the optic nerve into the eye and forward at the ora serrata. Although not adherent to the retina, it is firmly against it, all over the interior of the eye. Anteriorally, the lens rests against it and the zonular fibers which run from the lens back and outward to the ciliary body rest against it. The vitreous serves two functions. One, is to maintain the shape of the eye by keeping it full. The other is to be perfectly transparent and allow light to pass through the vitreous and on back to the retina.

The Visual Pathways

The visual stimulus passing from the retina back to the brain passes out through the optic nerve, into the cranial vault and into the optic chiasm where half of the fibers cross to join the other optic nerve, then back to a nerve center known as the lateral geniculate body, and from there on back to the extreme back part of the brain known as the occipital cortex.

The optic nerve starts where the nerve fibers leave the eyeball through the lamina cribosa and extends back across the orbit to the optic foramen. The optic nerve here is about an inch long and about an eighth of an inch across. It is S-shaped. This prevents it from being put on a stretch when the eyeball is rotated back and forth, leaving it free like a rope with slack in it. Like all other structures in the orbit, the ~~eyeball~~ optic nerve is covered with a connective tissue sheath. This connective tissue sheath is a direct continuation outward of the membrane which covers the brain. Supporting strands run inward from this nerve sheath into the nerve to support the nerve fibers and keep them separated, and to carry the small blood vessels in for nourishment. In the optic canal these fibers are very adherent both to the optic nerve and to the bone.

Inside the cranial vault the optic nerve extends for about a half inch where it joins the optic nerve from the opposite eye almost at right angles, forming what is called the optic chiasm. Here at the optic chiasm the nerve fibers from the nasal half of each retina cross over to the optic nerve of the other eye to join the nerve fibers from the temporal half of the retina of the opposite eye. The optic chiasm is situated just above the pituitary gland and is sometimes involved in tumors of this pituitary gland. When the nerve fibers from the eye leave the chiasm they are called the optic tracts. The left optic tract has the nerve fibers from the left side of the left eye and the left side of the right eye. The right optic tract has the nerve fibers from the right side of the right eye and the right side of the left eye. The optic tract goes backward to the nerve center called the lateral geniculate body. This is a relay station. From here the visual impulses pass backward to the occipital cortex.

Damage along this visual pathway causes a loss of a segment or all of

the field of vision depending upon the location of the damage. Thus when the optic nerve is cut, no visual impulses get through and the eye is blind. On the other hand, when the optic tract is cut or any place in back of it, the visual impulses from the half of the retina of each eye cannot get through and thus each eye is blind on that half of the visual field. As will be described later, studies of these visual fields help us to locate just where the damage has been done. By correlating these findings with other neurological findings lesions can be sometimes very accurately localized just as a telephone company by knowing which phones are dead can tell quite exactly where a pole is down on their lines.

Glaucoma

In your work you will frequently encounter patients blinded by glaucoma. The reported incidence of this disease varies, but it generally runs around 15% of the cases of reported blindness. It is a strange disease, characterized by an increase in intraocular tension, a characteristic gradual loss in visual fields, and an atrophy of the optic nerve with a deep depression or cupping of the nerve head. To understand the disease at all we must review the physiology of the intraocular fluids. These fluids are kept under a pressure to maintain the shape of the eye just as a balloon when blown up stays round, so when the eye has the fluids inside of it under pressure, it keeps the eye ballooned out nice and round. This fluid, or aqueous, comes into the eye from the ciliary body. The exact process by which the aqueous gets into the eye from the ciliary body has never been determined. Some men feel that it is an act of secretion, others think that it is formed by some type of an osmotic process. From the ciliary body the aqueous passes between the lens and the iris to the pupil and enters through the pupil into the anterior chamber. Some of this aqueous fluid is probably absorbed by the vessels on the surface of the iris. The rest of it passes to the angle of the eye, going through the spaces of Fontana into the canal of Schlemm and on out into the venous circulation. Like many of the body processes, this process stays in constant equilibrium. The pressure in an individual person will not vary over 4 millimeters of mercury. The average runs about 20 millimeters of mercury, with any tension between 13 and 29 millimeters of mercury being accepted as normal.

As has been said this process of maintaining intraocular pressure is in a delicate balance and when disturbed the pressure becomes either too low

or too high. The pressure being too low is a rare occurrence, and, unless associated with some diseased condition of the eye, is not of too great a significance. On the other hand a rise in the intraocular pressure is dangerous. When the pressure goes up the entrance of the optic nerve into the eye is disturbed and the nerve fibers atrophy either through direct pressure or from a disturbance in the circulation. At this point where the optic nerve leaves the eye, the thin, screenlike arrangement of the scleral coat, the lamina cribosa, which allows the nerve fibers through also constitutes a weakened area, so that the effects of the increased intraocular pressure are felt more here. When the pressure stays up these fibers gradually are stretched, giving way to form a bulge. The intraocular pressure can be raised by a process which increases the volume inside the eye, as a rapid engorgement of the blood vessels of the choroid and ciliary bodies, so that the fluid or aqueous can not drain off fast enough to allow for the increased volume. Or the direct process of fluid balance can be affected. The aqueous can be formed faster than it can be drained off or much more commonly, the angle is blocked preventing the aqueous from draining off as rapidly as it is formed. This blockage can occur in many ways. The lens can swell, pushing the ciliary bodies against the base of the cornea. The ciliary body itself, if it becomes swollen, will push forward against the cornea blocking the angle. Even wide dilatation of the iris causes the iris to become thicker as the radial fibers shorten up, and thus the iris presses against the angle. Inflammatory products may block the angle. Some angles are quite wide, others quite narrow. The narrower the angle the more susceptible it is to anything blocking it. In a great many of these eyes, the exact process by which the tension is raised, is not known, but it is generally felt the cause is a blockage at the angle. These eyes have either an unusually narrow angle or the

filtrating spaces there, the spaces of Fontana, are unusually small so that they are more easily blocked and with the gradual sclerosing process of age they block more readily than a normal eye would.

Classification and terminology used in these eyes is rather confusing at first, because the basic cause of glaucoma is so often not known. All glaucomas can be divided into primary glaucomas in which the cause is not known and secondary glaucomas in which the cause is known. The primary glaucomas in turn are divided into two main groups, the acute glaucomas and the non-acute, or glaucoma simplex, cases. There are other names for these two conditions, inflammatory and non-inflammatory glaucoma, incompensated and compensated glaucoma. In the terminal stage when the eye is blind and hard it is called absolute glaucoma.

Acute Primary Glaucoma

Clinical Picture. An attack of acute glaucoma is dramatic. It comes on rapidly. The eye is extremely painful. The pain may be so severe that it radiates over the head and even down on to the arm. At times the eye is not even suspected at first as being the site of the pain. There can be marked nausea and vomiting. The vision is rapidly reduced within a few hours going down to 20/70 or less, and within twenty-four hours almost to blindness. Generally just one eye is affected at a time. The lids are swollen. The conjunctiva is injected a bright red. The cornea has a steamy appearance like frosted glass. The anterior chamber is shallow. The iris markings look blurred. The pupil is dilated, and does not react to light. And, if it can be seen, the fundus picture shows the retinal vessels to be engorged and the disc hyperemic. The intraocular tension is markedly increased. The eyeball,

on palpation, feels like a marble under your fingers. And the tonometer tension will be well above 50.

To measure this intraocular pressure the doctor has his patient lie on his back and look either toward the ceiling or he has the patient hold his thumb at arm's length directly over his eye and look at it. The cornea is anesthetized with a medicine, usually pontocaine, and a delicate instrument placed gently upon the cornea. This instrument has a little plunger running up the middle of it which moves a long lever. The more the pressure inside the eye, the higher this plunger is pushed up and the higher the reading on the instrument. This instrument is called a tonometer.

Pathology. The picture of acute glaucoma is a picture of congestion and edema. All of the vessels of the eye are markedly engorged, and the surrounding tissues are edematous. The ciliary body is swollen from the edema, the iris is swollen and together they block the chamber angle. So, the worse the inflammation the more the blockage of the angle, creating a vicious circle. Also the pressure as it goes up paralyzes the pupil so that it cannot contract down to pull the iris away from the chamber angle. The corneal epitheleum is edematous like the rest of the eye. The edema spoils the transparency of the cornea and thus prevents looking into the eye.

Etiology. The exact cause of acute glaucoma is not known. There are certain predisposing factors. It is known to be hereditary. Several members of a family often will show up with the condition. A study of the age of these patients shows it to be uncommon before 35 but of increasing frequency after that. Most of these patients are hyperopic with an abnormally small eye. Also, most of these patients show an instability of the vasomotor system, i.e. their heart and blood vessels respond more rapidly and more completely to any emotional stress. An attack of acute glaucoma is often found to follow some emotional crisis.

Diagnosis. A history of sudden pain in the eye with marked loss of vision and the examination showing the acute conjestion, cloudy cornea, a large fixed pupil, and increased intraocular tension point definitely to an acute glaucoma.

Treatment. The treatments used in this condition are non-surgical and surgical. In the non-surgical treatment medicines are used to alleviate the pain and to bring down the tension. To alleviate the pain, morphine is generally used. To bring down the tension, drops or ointment containing some medicine to make the pupils small and thus pull the iris away from the angle and allow the fluid to escape through the angle, are used. These medicines are generally pilocarpine in a $1\frac{1}{2}$ or 2% solution and eserine in a 1% or 2% solution. Ice packs are used locally on the eye to relieve pain and conjestion. A saline purge is given to dehydrate the body and thus pull the fluids out of the eye and into the bloodstream. And also injections are sometimes given into the blood of a hypertonic solution such as 50% glucose to draw the fluid into the blood vessels of the eye and on out of the eye. If these measures succeed, the angle of the eye is opened, the aqueous is drawn out of the eye both into the circulating blood by the hypertonic solutions and out through the angle of the eye by its normal pathway. The pressure goes on down, the inflammation then begins to subside, and the eye is saved.

If these measures do not succeed within 24 to 48 hours operative interference is necessary. The operation generally performed on these eyes in an acute episode of glaucoma is called an iridectomy. In this operation, a small incision is made at the corneal-scleral junction into the anterior chamber. With a fine forceps the surgeon reaches in, takes a hold on the iris and draws it out through the wound. He makes one radial cut which extends from the pupil border to the base of the iris as far as he can get. Then, still holding on

to the iris, he pulls it to one side tearing the iris loose at its base, over the extent of the wound. This piece of iris which has been torn loose is cut away by cutting again from the pupil edge to the base to the iris. This removes a segment of the iris diaphragm which can be compared to cutting out a piece of pie. By tearing the iris loose at its base, the angle is opened widely. The edges of the remaining iris are replaced carefully and the wound is closed. The results of this operation are usually very good, the tension of course is reduced to normal by opening the eye and it generally remains normal. Vision returns as a rule within a short time.

Subacute Primary Glaucoma

Glaucoma can also occur in a similar but milder form. The degree of severity may vary from what has been previously described under acute primary glaucoma down to mild episodes of discomfort and slight redness of the eye which are hardly noticed by the patient. The pathology here is a milder picture of the acute glaucoma. The etiology is the same. The diagnosis is sometimes quite difficult. When the picture is severe enough to bring the patient to the doctor the diagnosis is not often difficult. However, in the routine history the story is sometimes rather baffling and the doctor has to take diagnostic steps exactly as will be described under the next heading, glaucoma simplex. The treatment is as described for the acute attacks. Medical measures are tried first. If the tension can be controlled in this way, nothing further is necessary. If not, some type of filtering operation is necessary to allow the fluids to escape from the eye and maintain a normal intraocular tension.

Glaucoma Simplex or Chronic Primary Glaucoma

This condition of chronic primary glaucoma will be found much more often than acute glaucoma. Whereas acute glaucoma presents a dramatic episode, chronic primary glaucoma shows a very silent insidious onset. There is no pain, although some patients occasionally have a faint dull aching over the eyes. Vision is very gradually lost. This loss of vision starts in the peripheral field, not in the central field where it would be noticed. You remember the discussion of testing of vision. There was a description of testing visual fields. It was explained there that the visual acuity for central vision was very high, but that the visual acuity rapidly dropped as the field was tested away from the point of fixation. However, the vision for more gross form remained present out about 90 degrees. It was also explained that the visual field was really a map of the retina with the center being the point of fixation and the blind spot being the entrance of the optic nerve. Now, with these patients, the vision, as it is being gradually lost in the peripheral fields remains good in the central field. And, because of the overlapping of the two visual fields, the loss goes unnoticed until it approaches the central area. The eyes show no evidence of inflammation, so that on external examination they appear quite normal. On examining the patients with an ophthalmalscope, the ophthalmologist finds the nerve head does not have a normal appearance. Instead of having a small normal physiological cupping, the cupping is larger. The nerve head itself looks whiter. There are no small capillaries visible if it is far advanced. And, if it is far advanced, this cupping has extended to the point that the vessels, instead of appearing to go straight down to the lamina cabrosa, turn back on themselves out of sight behind the choroid and retina and then reappear again at the bottom of the cup. The intraocular

tension is variable. It may be within normal limits at some times, or it may go as high as 30 to 40 mm. of mercury. Generally both eyes are involved, with one more advanced than the other.

Pathology. Here, the picture instead of being one of congestion and edema shows the results of the increased pressure. The cornea generally appears normal. The angle shows the iris brought forward against the cornea with adhesions so that the normal angle has been obliterated and the fluid thus cannot go out its normal pathway through the spaces of Fontana and into Schlemm's canal. The iris is generally atrophic. The ciliary body shows atrophy with deposits of colagenous material. This material will appear in practically any tissue with degenerative changes. Under the microscope it just looks a uniform pink color. The optic disc shows the nerve fibers to be atrophic. The lamina has very few fibers above it so that the physiological depression instead of appearing as if the vitreous dipped down into a little funnel into the nerve head now is pushed down and out like a small blister. The lamina vitrea does not give at all to this increased pressure, but the optic nerve head outside it is pushed outward in all directions. Thus, under the microscope, it appears as if the vitreous had pushed all of the nerve tissue back through the hole in the lamina vitrea. Then in back, where the resistance to the pressure is not so high, it looks as if the vitreous had bulged further in all directions back behind the lamina vitrea pushing aside the choroid and the scleral coat. There is also an atrophy of the optic nerve for a way back.

Etiology. As with acute glaucoma, there is frequently a family history of glaucoma. The disease occurs with an increasing incidence after the age of 40. It is much more frequent in hyperopic eyes. Also, there are more signs of senile degeneration. The drainage system shows more evidence of damage, the chamber angle being blocked by adhesions between the iris and the cornea.

Diagnosis. The differential diagnosis of chronic glaucoma from senile cataract is important, so that the patient is not told "Well, you have cataracts but your vision is not bad enough for an operation, so just wait until they get worse and then we will operate." Here, if the patient also has a glaucoma, the vision goes down, but no operation can bring it back. More important than differentiating chronic simple glaucoma from cataract or other conditions which cause a diminished vision, is the diagnosis of the condition itself, early, while the patient still thinks he has a normal pair of eyes. The responsibility here is upon the ophthalmologist in doing his routine eye examination for glasses. As part of his refraction routine he takes a history of any episodes of diminished vision or halos around the lights at night, these halos being caused by the slight corneal edema when the pressure is slightly elevated. In his fundus examination he notes the condition of the optic nerve head, and if the physiological cup appears larger than normal he checks further. He routinely will check the intraocular tension on all patients over 45. If any of these examinations show suspicious findings then the ophthalmologist goes further in an effort to establish a diagnosis. He will check the visual fields to see if there are any early signs of field loss in the peripheral fields. He will run what are known as provocative tests on these patients. These provocative tests are attempts to embarrass the balance of the intraocular fluid. The tests include a drinking test in which the patient is seen in the morning. He has taken no fluids since 10 o'clock the night before. His intraocular tension is checked and he then drinks a quart of water rapidly. His tension is then checked again at twenty minute periods for an hour. This water, on an empty stomach, rapidly goes into the blood stream, and dilutes it down. This dilute blood, as it flows through the eye, allows fluid to get into the eye more rapidly. Then, if the angle of the eye does not carry this aqueous fluid

off as rapidly, the tension goes up and this rise is found on the repeated tonometer checks. Another test is known as the dark room test. Here the patient's tension is checked and then he sits in the dark room for an hour. In the dark, his pupils dilate and the dilated pupil blocks the angle of the eye. These two tests, the drinking test and the dark room test, are often combined.

There are other tests which are less reliable but sometimes used, such as the caffeine test where the patient drinks coffee, in the belief that caffeine also stimulates secretion into the eye. The vasomotor test in which the patient's tension is taken, then his hand is dipped below the wrist in a dish of ice cold water and his tension again taken. This will sometimes cause a rise in the intraocular tension. Another test is to put a blood pressure cuff around the neck of the patient and then raise the pressure in the cuff to halfway between what are known as the systolic and diastolic pressures. There is no danger to this but it causes a venous congestion in the head by blocking somewhat the venous return. This causes a rise in the intraocular pressure, which is found by checking the intraocular tension. Another test is known as the mydriatic test in which a mild mydriatic is put in the eye. The mydriatic is a drug which dilates the pupil and thus blocks the chamber angle. By checking the pressure before and after dilatation a rise in tension may be found. This is not too commonly used as some men feel that once the pressure starts going up from a blockage it cannot be relieved quickly enough.

Treatment. Here again the treatment is first medical and then, if this fails, surgical.

It must be emphasized here that in the treatment of glaucoma simplex the vision which has been lost cannot be regained. All efforts of treatment are directed toward maintaining the intraocular tension at normal so that there

will not be further loss of vision. This is important, as often times the patient becomes discouraged feeling that nothing is gained and discontinues his treatment, only to lose more vision.

The medical treatment is the same as for the acute and subacute glaucomas. Drugs are used which pull the pupil down and make it small, myotics, and thus open up the chamber angle allowing the aqueous to escape more freely. The drugs most commonly used for this are pilocarpine and eserine. The pilocarpine is generally in the form of a solution to be used as drops and the eserine is generally in the form of an ointment. There are newer drugs on the market, some of which have a longer lasting effect, but these are not yet in general use. A patient using medication like this must also limit his fluid intake. Fluids should not be limited to the point of being thirsty, but he should never drink excessive amounts such as ice tea, glass after glass, in the summertime. He should avoid emotional upsets when possible and if they do occur he should apply another drop of the medicine in his eye. When he goes into dark rooms, such as movies, he should put another extra drop of the medicine in his eyes. He should avoid wearing tight collars as they will cause a congestion in the head and in the eyes and thus raise the pressure in the eyes. And he must return to his ophthalmologist for regular checkups to make sure that his glaucoma is staying under control.

When these routine checkups show that the vision is failing and that the tension is not staying under control, the ophthalmologist considers some form of surgical intervention. All forms of surgery for glaucoma are aimed at making a new pathway for the aqueous to get out of the eye. There are several types of operation used, all with the same goal. One, known as the corneoscleral trephine is performed by lifting down a fold of conjunctiva from above to the edge of the cornea and then drilling a small hole into the anterior

chamber through the junction of the cornea and the sclera. Then the conjunctiva is sewed back leaving a pathway from the anterior chamber through the small hole where the button of tissue was removed for the aqueous to escape. Another operation is known as iridencleisis. Here a flap of conjunctiva is also made as for the corneoscleral trephine. Then a small incision is made into the anterior chamber, starting just in back of the limbus. Through this opening the iris is drawn out and a radial cut made in it. Then one of the pieces from this cut is folded back into the wound and the wound closed. The iris tissue then acts as a wick, allowing the aqueous to escape from the anterior chamber, through the wound and into the subconjunctival tissue where it is absorbed. A third operation is known as cyclodialysis. Here a conjunctival flap is made and a small opening then made into the sclera about five or six mm. back from the limbus. A small flat instrument, similar to a spatula used in cooking, is then slipped carefully between the ciliary body and the sclera forward into the anterior chamber and then moved from side to side. This opens up the chamber angle so that the aqueous can pass out into the potential space between the sclera and the choroid, where it is absorbed by the choroidal vessels.

Congenital Glaucoma - Buphthalmos

This form of glaucoma occurs during the first year of life. The parents first notice that the cornea has a whitish appearance. On examination by the ophthalmologist, he finds that the cornea has a cloudy appearance, the cornea is larger than normal, the child generally has defective vision and the eye sometimes is injected. In taking the tension the eye generally feels hard and the tension by tonometer is quite high, 50 to 75 mm. of mercury.

Pathology. The pathology here is a failure of development of the eye. During fetal life, the anterior chamber as it develops, starts toward the center and spreads lateralward, as a separation between what will be the iris stroma and the cornea. In these eyes, the separation has not gone all the way back to the canal of Schlemm. The angle in these eyes instead of being sharp is quite broad, and there is considerable tissue between the chamber and the canal. This prevents the aqueous from escaping from the eye, but the aqueous continues to be formed and the pressure builds up. Then, the sequences of increased intraocular tension follow.

Treatment. The only effective treatment in these eyes is surgery. Iridencleisis is sometimes used. More recently, an operation known as goniotomy has been employed. In this, the surgeon enters a knife into the cornea just inside the limbus, passes it across the anterior chamber, and cuts through the remaining tissue along the cornea in the angle to the canal of Schlemm. He then swings his knife along the angle, breaking down the fine adhesion-like tissues in the angle. Thus there is left a pathway for the aqueous to get out of the eye.

Secondary Glaucoma

Secondary glaucoma includes all forms of glaucoma where the cause is known. The name means that it is secondary to some known cause. Anything which will block the chamber angle and prevent the escape of the aqueous and thus cause an increase in the intraocular tension can cause a glaucoma.

Iridocyclitis. An inflammation inside the eye in the acute stages causes an increase in pressure by blocking the angle with inflammatory products, and through a swelling of the ciliary body. Later on, the iris which is pushed up against the cornea may become adherent to the cornea from the scarring

caused by the inflammation. Also, in iritis adhesions are formed between the edge of the pupil and the lens. If these adhesions extend all of the way around the pupil, or across the pupil, the aqueous cannot get from behind the iris, where it is formed by the ciliary body, forward into the anterior chamber to leave the eye through the angle. The iris is bulged forward and the tension builds up behind. Also as the iris is pushed forward by this fluid it is pushed against the cornea, blocking the angle of the anterior chamber, thus there is a double blockage to the exit of the aqueous.

Treatment. During the acute stages, the treatment is that of the iritis. If the patient's tension remains high, the doctor sometimes makes a small knife opening into the anterior chamber to drain off the aqueous and lower the pressure temporarily. This is called a paracentesis. When the iris bulges forward, the tension can sometimes be lowered by inserting a knife through the cornea, then into the iris, through the bulging part and then out again through the other side of the bulge, making two puncture holes in the iris to allow the aqueous to escape. Sometimes an iridectomy is necessary. Here a conjunctival flap is made, a small opening then made into the anterior chamber and a piece of the iris brought into the wound and cut out. This leaves a defect in the iris much like when the first piece of pie is removed from the pie. The exact way in which this relieves the rise in tension is not known, but following an iridectomy, the tension often returns to normal and stays there.

Tramatic cataract. When the lens is injured breaking the capsule and allowing the cortex to be exposed to the aqueous, the cortex often swells rapidly. As it rapidly swells it presses the iris forward against the cornea and blocks the exit of the aqueous. The treatment is operation to remove the lens material.

Other types of secondary glaucoma include those coming after the ex-

traction of senile cataract. Here the angle is blocked either by remnants of the lens capsule or pieces of iris, or by postoperative iridocyclitis, or it is blocked by adhesions between the iris and the cornea which are formed while the angle is collapsed following the surgery. The treatment in these cases is often difficult as they do not respond well to medication. The operation generally used here is a cyclodialysis. This was described under glaucoma simplex. Intraocular tumors will cause a rise in tension both by mechanically blocking the angle with fragments of the tumor which pass forward and start growing, and by the tumor mass itself pressing forward and pushing the iris up against the cornea. Following a thrombosis of the central retinal vein there is often a rise in the intraocular tension. Treatment in these cases is very difficult.

Absolute Glaucoma

This term, absolute glaucoma, is used for a blind, hard eye. It can follow any of the previously described glaucomas. On examination the eye is found to be blind, the cornea is cloudy, the eye when you feel it feels like a marble under the lids, the tension is very high. Some of these eyes are painful, sometimes extremely painful. At other times they are quite insensitive.

Treatment. If the eye is painful it should be removed, if medical treatment cannot control the pain. The ophthalmologist is always suspicious when the patient is found to have an absolute glaucoma in one eye and the other eye normal. He cannot look inside this glaucomatous eye to look for the cause, so he wonders whether or not there is a tumor in the eye. Because of this the ophthalmologist will often recommend the removal of a blind eye even though the patient would rather retain it.

THE DIFFERENCE BETWEEN ETIOLOGY AND EYE CONDITION

In the reports which must reach the social worker, in order that he may properly evaluate the status of his client, he will find a listing of both the etiology and the disease condition of the eyes. The etiology refers to the known cause of the patient's difficulty, while the diagnoses may be merely a statement of the end results. For example, a patient with central nervous system syphilis may have severe loss of vision. Eye examination reveals an advanced primary atrophy of the optic nerve. In this case the etiology, or cause, is the syphilis of the central nervous system, although the condition found is listed as optic atrophy. In all cases where the cause can be assigned, either by certain laboratory tests or by other examinations of the patient, the etiology is established and can be listed in the report. In many cases, however, no causative agent can be ascribed and in such instances no etiology can be stated. In these particular cases the term "unknown to science" may be used.

In general etiologies may fall under certain broad groupings. These include infection with specific organisms, traumatic injury, degenerations--either on a hereditary or aging basis, tumors of various sorts, certain generalized systemic diseases of the body, developmental abnormalities, which may or may not be hereditary, and others. The example of central nervous system syphilis will serve to demonstrate a disease condition (primary optic atrophy) due to a specific organism (the spirochete of syphilis). As an instance of degeneration we may point out macular changes in which the central area of the retina is damaged. Such a patient loses his central vision. If a definite family history can be elicited such a change of the retina can be shown to be hereditary. In other instances, in older people, the same change may be merely due to aging in the eye. The

example of a blow to the eye with resulting visual loss needs no further explanation. One of the systemic diseases which produces visual changes is diabetes mellitus. This generalized metabolic disease may reduce vision because of the formation of hemorrhages in the retina, mainly in the region of the macula--the central area of vision. A malignant tumor of the eye may require removal of the globe with resultant loss of vision. In the case of developmental abnormalities it may be possible in some cases to assign a hereditary basis, but in others it will be impossible. Interference with growth of a certain part of the ocular system may occur during development, resulting in severe abnormalities.

There remain, however, many ocular conditions which have no distinct cause. The majority of cases of glaucoma fall into this group. The usual senile cataract is another example. Doubtless, with the advance of knowledge, this group will be gradually narrowed until more and more causes of disease conditions in the eyes will be definitely established.

To recapitulate, the etiology of the particular patient's difficulty is the cause, the eye condition is the effect.

DISEASES OF THE IRIS AND CILIARY BODY

The iris is the opaque diaphragm of the camera of the eye which allows changes in size of the pupil. It is a highly vascular structure with muscle tissue present to allow for continuing changes in the pupillary opening. Certain abnormalities of the iris, some of which are hereditary, may result in interference with vision. There may be an abnormal positioning of the pupil (ectopia of the pupil) or multiple pupillary openings (polycoria). A sector of the iris may be entirely absent giving the pupil the appearance of a keyhole. This defect is known as a coloboma and is often associated with a similar defect in the choroid and retina which diminishes visual acuity. Certain individuals will show absence of the iris or aniridia as it is called. The inevitable result in such cases is an intractable glaucoma with gradual destruction of vision. One further condition should be mentioned. This is a degenerative process known as essential atrophy of the iris in which for unknown reasons there will be a gradual disintegration of the iris tissue of a patchy nature. This gives a peculiar moth-eaten appearance to the iris and an associated overgrowth of tissue into the filtering angle of the anterior chamber results in a glaucoma which is very difficult to control. In many instances it is possible to prove, by careful family study, that essential iris atrophy is hereditary in nature.

Inflammation of the iris is known as iritis. This term is commonly used, although in most cases the ciliary body is also involved and to be technically correct the term should be iridocyclitis to designate inflammation of both structures. If the iris, ciliary body and choroid are involved, a uveitis is said to be present, since all three components of the uveal tract are affected. Since the iris and ciliary body are richly vascular it is possible for any blood-borne infectious process to reach them and to set

up an inflammatory reaction. Such cases of iritis due to a specific organism are rare, however. The vast majority of affected individuals have no assignable cause for their difficulty. It is thought by some workers that such cases are due to local sensitivity to bacterial products arising elsewhere in the body.

When inflammation of the iris occurs, there results an engorgement of the many blood vessels and an actual swelling of the iris tissue. This prevents adequate movement of the pupil and it may be extremely difficult to make it larger by the use of drugs. The patient complains of pain which may be of an aching nature and worse at night when he lies down. Involvement of the ciliary body is shown by the blurring of vision which results from weakening of the action of the ciliary muscle in focusing the eye. The inflammatory response in the iris tissue results in an outpouring of cells and other blood material into the aqueous fluid of the anterior chamber, producing cloudiness of this watery medium. Thus, when a fine beam of light is directed through the aqueous, it intercepts the material suspended in the fluid and becomes visible. This phenomenon is very important in diagnosis of the milder cases. The floating cells and inflammatory material in the aqueous fluid often become deposited on the back surface of the cornea to produce masses of material known as keratitic precipitates. These further interfere with vision.

The greatest danger in iritis is that the inflamed iris may become adherent to the lens which lies just behind it. This is prevented by the use of drugs which dilate the pupil to pull it away from the central protuberant area of the lens. The main drug used is atropine. This also paralyzes the ciliary muscle tissue and puts it at rest, thus eliminating a source of pain.

If the iris is allowed to become adherent to the lens a chain of

events starts which may lead to loss of vision. The adhesions between the two structures are called posterior synechia. They may occur in various places along the border of the pupil and produce an irregular pupil when atropine is used to dilate the iris. Such synechia, if unbroken, tend to make the iritis chronic. If the synechia are complete, or around the entire pupillary border, blockage of the pupil ensues and the aqueous can no longer flow forward into the anterior chamber. The continued aqueous production by the ciliary processes balloons the iris forward except where it is held in place at the pupil border and at the iris base, resulting in iris bombee. This ballooning iris then presses into the filtering angle of the anterior chamber blocking drainage of the aqueous fluid from the eye. Thus a severe glaucoma is set up, since the aqueous continues to be formed. Relief in such a case can be gained only by surgery.

In other cases there may be an inflammatory membrane laid down over the anterior lens surface within the pupil in addition to the posterior synechia. This membrane blocks vision and cannot be removed by surgery.

Since the lens of the eye is nourished entirely by way of the aqueous fluid the change in the composition of this fluid during iritis may interfere with the lens metabolism. In such a case the lens is likely to become opaque, particularly in its posterior layers, resulting in the formation of a cataract.

These various complications of iritis can be prevented in many cases by early vigorous treatment and dilatation of the pupil with appropriate drugs is the most important local measure. In addition, the patient is subjected to a thorough physical examination in order to remove any remote causes which may result in iritis in the eye. Since iritis can be due to syphilis a blood test must be done. Any infection in the body such as abscessed teeth, infected tonsils and so forth must be remedied. A parti-

cularly resistant form of iritis may be due to tuberculosis or to undulant fever. In the great majority of cases, however, no other causative agent can be found after thorough examination. Iritis is likely to recur and in some instances its onset appears to be seasonal.

The remaining cause of loss of vision from disease of the iris and ciliary body is much less common. This is the formation of malignant tumors in these tissues. Such tumors will be discussed at length later, but in most cases their discovery demands removal of the involved eye in order to protect the patient from spread of the process which may cost him his life.

DISEASES OF THE CRYSTALLINE LENS

Since the crystalline lens is necessary for focusing light on the retina, any disturbance in its structure, position or transparency will result in decreased vision. If loss of transparency occurs the resulting change is known as a cataract. Such a lens change may be localized to certain areas only or it may involve the entire structure.

There is a large group of so-called congenital lens opacities which are present either at birth or which progress during the early years of life. No assignable cause can be stated for many of these cataracts, but more recently there has been more light shed on their origin. For example, it has been found that when a mother suffers from German measles during the first three months of her pregnancy there is likely to be a centrally located cataract in the new-born baby. In addition there may be deafness, various abnormalities of the heart and mental deficiency. The toxic agent appears to act on the structures of the embryo which are growing the most rapidly at the time and the lens of the eye is among these. This fact opens new fields for study in the etiology of congenital cataract and other causes may be found. Certain other congenital cataracts are thought to be due to an abnormality in the calcium and phosphorous content of the body in the developing child. This results in a condition known as tetany in which there may be muscular spasms. There appears to be a relationship between a deficiency of vitamin D and this condition. Thus study in the field of nutrition, both of the pregnant mother and of the baby, may aid in the prevention of such cataracts.

Certain of the developmental cataracts are definitely known to be hereditary, being passed down through a family in various patterns. It is possible that additional study of heredity in cataract will give us a further tool in prevention of blindness.

Regardless of cause the group of congenital cataracts have a relatively poor outlook with treatment. In many instances there are other abnormalities in the patient's eyes. Vision may never develop satisfactorily either because of lack of development of the central area of the retina or because it never has an opportunity to see clearly because of the opacities in the lens. Many children will have a continuous roving movement of the eyes (nystagmus). In such instances successful removal of the cataract seldom results in much visual improvement.

The majority of cases of cataract occur in the older age groups and are known as senile cataracts. There is no definite cause for these other than the aging process. A patient with such a cataract will note a gradual decrease in vision with no associated pain or other symptoms. He may first find that glasses need to be changed frequently due to increasing density of the lens of the eye. Such an increase in density makes the eye more near sighted and the patient may find that he can now read without his glasses. This is known as "second sight." The process may remain stationary for a varying period of time in different patients. In such a case the use of a particular medication or treatment may be claimed as the reason. Nevertheless, there has been no successful medical treatment discovered to date and the greater number of cases gradually progress until surgery is necessary.

The surgical treatment of cataracts consists of removing the cloudy lens from the eye by various means. Following operation the patient must always wear glasses in order to see, since the power of the lens of the eye must be substituted for by a corrective lens. Since the crystalline lens is absent no focusing of the eye is possible and bifocal glasses are necessary, regardless of the patient's age. In the senile cataract group surgery is usually successful in one operation, since the entire lens is removed together with its outer envelope, the lens capsule. Such an operation is

termed an intracapsular cataract extraction. In certain older people it may be necessary to remove the lens from its capsule, leaving the outer lens envelope behind. This is known as an extracapsular cataract extraction. About 15% of such cases will undergo a further opacification of the retained capsule, producing a secondary cataract. This must be treated by further surgery, in which an opening is cut in the cloudy capsule in order to produce a clear area for the passage of light. Such a secondary procedure is known as a needling (or discission) of the membrane.

In earlier days all surgery in the older age group was of the extracapsular type and it was thought necessary for the lens to become completely cloudy before surgery could be done. This was known as "ripening" of the cataract and in such cases the lens material separated well from the capsule, which was left behind. More recently the intracapsular type of operation has made prolonged blindness unnecessary and the patient can have his cataract removed as soon as his vision falls below a useful level. It is useless, however, for an operation to be performed on one eye alone if the second eye is still seeing well, since an eye without a lens (aphakic eye) will not function well with a normal eye. This is because the necessary glasses after cataract removal produce magnification and the size of images in the two eyes will be different.

It is most important in the older age groups in which cataract is suspected as the cause of quiet, progressive loss of vision to rule out other conditions which might produce a similar situation. Chief among these is chronic glaucoma, which may progress without pain or other symptoms. Next in importance is degeneration of the retina, particularly in the central area, which also occurs without pain. The diagnosis of cataract is made too many times on insufficient evidence and in the meantime the patient gradually loses his sight through other causes, none of which can be remedied to the

extent that senile cataract can. Therefore, any case of early cataract should be kept under fairly frequent observation, since the presence of other disease conditions must be searched for at a stage when the retina can still be visualized through the gradually clouding crystalline lens.

The results in cataract extraction in the senile age group are generally good. About 75% of cases are able to read the newspaper with their glasses by three months after operation. This time interval is necessary before final fitting of bifocal glasses, since the eye changes its shape gradually during the process of healing. Many surgeons give "temporary" glasses immediately after leaving the hospital in order to aid the patient during this waiting period. There is a definite surgical risk in such surgery, although it is small enough to be no deterrent to the operation. About 1 eye in 200, or 0.5%, are lost through various operative and postoperative complications.

The age of the patient makes little difference in the feasibility of surgery. Cataract operations have been successful with patients well into the 90s. Provided the patient is able to lie quietly in bed for several days after surgery, he will be a suitable candidate. The operation is usually done under local anesthesia, depending of course upon the surgeon's judgment, and is no more difficult for the patient to withstand than a tooth extraction.

In children and in younger adults the one stage operation is not as feasible because the zonular fibers which hold the lens in place do not rupture as easily. In these individuals multiple operations are usually done. In very small children multiple needling operations may be sufficient, since by making an opening in the lens capsule the aqueous is able to dissolve the lens material and carry it away. In others a needling may be followed by rapid ripening of the lens material which then swells and produces glaucoma.

In such cases it may be necessary to open the eyeball and wash out the mature lens material. Such a procedure is known as a linear cataract extraction. The possibility of such lens swelling after needling may occur in any age group and such patients must be kept under frequent observation.

There is one further group of cataracts which should be mentioned. Such are known as complicated cataracts and may be the result either of inflammation in the eye or of metabolic or degenerative disease. Certain metabolic diseases such as diabetes, absence of the parathyroid glands after too extensive removal of the thyroid, generalized skin disease and certain muscular disorders may produce cataractous changes in the lens of the eye. Retinitis pigmentosa may have associated cataracts. Poisoning with certain substances may cause lens clouding. In any event such lens changes appear due to interference with the normal nutrition of the lens. The outcome of operation in such eyes depends upon whether other eye damage is present.

Traumatic cataracts need only to be mentioned. Any injury which damages the capsule of the lens produces the same result as needling. The lens material in direct contact with the aqueous becomes cloudy. In any individual with such a cataract operation for vision is not indicated since the fellow eye will not act with the operated eye for reasons stated above. In some cases surgery will be necessary for other reasons, such as glaucoma due to the lens swelling.

In certain individuals another lens difficulty may be present. The zonular fibers which hold the lens in place may be deficient, either in certain areas or completely. This allows the crystalline lens to be displaced from its central position or to lie entirely free within the eye. Such a condition is known as dislocation of the lens. It is congenital in many cases and in certain of these it is known to be hereditary. In some it is associated with other bodily abnormalities. One of these is a condition

known as Marfan's syndrome, in which dislocation of the lenses of both eyes is accompanied by an extremely elongated body habitus. These individuals have extremely long bones, are tall and thin and their hands and feet are elongated. This condition of the hands and feet is known as arachnodactylia, or "spider fingers."

With a partial dislocation of the lens vision may be very poor, since light passes through the curved side of the lens rather than its center. In some cases there may be a clear area in the pupil alongside the lens where refraction can be successfully done with glasses similar to those used after cataract removal. In other individuals removal of the lens may be necessary in order to give the best vision.

When the lens is completely dislocated it may lie in the posterior portion of the eye behind the iris. Vision may be improved by glasses, but the lens itself may cause further complications. In some cases it will continually irritate the area where it lies and produce either inflammation or glaucoma. Rarely it may fall forward through the pupil and become caught in the anterior chamber leading to a severe glaucoma. In such a patient the lens must be removed surgically and the results are many times disappointing. If danger of the lens dislocating forward through the pupil is present the patient is many times given drugs to use at bedtime in order to keep the pupil small during the hours of sleep, when he may be lying on his face.

Regardless of the complications present in diseases of the lens, this group of patients has the best outlook for restoration of vision by present methods of treatment. Particularly in the older age group the possibilities for rehabilitation are tremendous.

DISEASES OF THE CHOROID AND RETINA

The light-sensitive retina, the lining membrane of the posterior segment of the eyeball and the vascular choroid coat, which immediately underlies the retina, are usually involved together in disease changes. The injurious process may start in either the choroid or retina, but because they are in direct contact with each other rather quickly both layers are damaged. Any injury to them results in a varying loss of function of the retina at that spot, producing a corresponding blind area in the field of vision. Once damaged the retina has little power of recovery and the blind area tends to be permanent. If the damage occurs in the central area of the retina and choroid, the macula, there will be a loss of central vision with a central scotoma in the visual field. This permanently lowers the visual acuity and the amount of loss corresponds to the size of the area blinded. If large enough the blind area may cover even the large "E" at the top of the visual test chart and the patient will then have less than 20/200 vision. In other instances the spot of choroid and retina involved may be away from the central visual area and the patient may be entirely unaware of the disease process, since there is no effect on the central reading vision.

Inflammation of the choroid and retina is known as chorioretinitis. In general it is due to the same diseases as were listed for the causes of iritis. Syphilis, tuberculosis and undulant fever are among the main causes of the chronic types. Direct bacterial infection by way of the richly vascular choroid may result in a very severe type of chorioretinitis, since the germs gain entrance to the vitreous space and may form an abscess within the eye, with complete destruction of the ocular function. In many cases the cause is as obscure as in iritis, with no bacteria found within the diseased areas. Presumably such a chorioretinitis is due to local sensitivity to

bacterial products from elsewhere in the body and thus other sources of infection must be searched for. Even if successfully treated it must be repeated that the areas involved heal with destruction of that portion of the retina and permanent blindness of a corresponding part of the visual field results.

Certain congenital defects of the choroid may be the cause of poor vision. In some instances they are hereditary. Coloboma of the choroid is the most common, with a defect in the choroid below usually extending to the region of the optic nerve. Since the choroid does not develop, the retina over it is retarded and abnormal due to lack of vascular supply. In most cases the central vision is poor and in others the coloboma may involve the macula itself, interfering completely with central vision. Choroideremia is a rarer abnormality in which there is almost complete absence of the choroid, with resultant lack of function of the overlying retina. The greater involvement of the periphery of the retina results in night-blindness, while in most cases there may be a small central patch of normal choroid and retina. This results in fairly good central vision but with a marked constriction of the field of vision.

Tumors of the choroid occur and, since they are of the pigmented variety in most cases, are extremely dangerous to the patient's life because of their great tendency to spread to other parts of the body. The only treatment is early removal of the affected eye. Such new growths will be discussed at greater length below.

The greater proportion of eyes with retinal involvement which destroys vision are affected by inflammation. In most cases both the retina and choroid are involved and the better term would be chorioretinitis. This has been discussed above.

A second major cause of retinal damage is various degenerative processes where the retina is destroyed wholly or in part. Foremost among such conditions is primary retinal pigmentary degeneration or retinitis pigmentosa. In many such cases the disease is found to be hereditary and in others an inherited basis for the disease can be presumed. The first symptom in many of these affected individuals is night-blindness. This is because the process starts first in the periphery of the retina where there is a greater number of rods rather than cones acting as visual receptors. The patient may have difficulty in getting about in twilight; for example, a child will not be able to play with the neighborhood children after sunset because he cannot see well enough. The characteristic picture seen by the ophthalmologist in the retina consists of peculiar deposits of dark pigment in the periphery of the retina which gradually may approach the central macular area over a period of time. The optic nerve becomes waxy-looking and the blood vessels of the retina are narrowed. The patient may retain good vision as far as testing is concerned, because the central area of the retina is not involved, but his field of vision will be so narrowed that he has difficulty in getting about. A further complication in certain cases is the formation of a cataract which lies in the posterior central area of the lens, decreasing vision even more. In selected cases the lens can be removed successfully in order to aid vision, provided the central area is not yet involved. The disease, retinitis pigmentosa, exists in many varieties so far as the order of degenerative changes occur. The typical case has been described above, but in others the macular region may be involved early to destroy central vision. In others the disease may progress to a certain point and then stop. It has been found that the disease picture varies with the family studied and that the different varieties hold fairly true for a given pattern of inheritance. Contrary to the usual opinion the disease may

be inherited in patterns other than the purely recessive one.

There has been found no treatment of value for primary pigmentary retinal degeneration up to the present time. Many things have been tried without conclusive results. The recent flurry over the tissue-therapy method used by Filatov in Russia has not been substantiated by work in this country. It must be remembered that social position, income and prestige in Russia are governed by the production of glowing results.

An even more common variety of degeneration in the retina which produces loss of vision is that involving the macula or central region of the retina. It will be recalled that this area is the point where the more acute reading vision is perceived. Many types of macular degeneration have been recognized. There are those which occur in children or young adults. In many of these cases a definite hereditary pattern can be established and in some the onset of blindness will occur at a certain age in all instances. In adults and old people degeneration of the macula can be proved to be inherited in some cases, but in many it cannot. This produces a painless gradual loss of vision which can easily be confused with the formation of cataracts or with the quiet, painless chronic glaucomas. If such a change is present behind a dense cataract it may not be discovered until after the lens has been removed and a disappointing visual result ensues. The loss of central vision in older people is a distinct disability, since many of their pleasures involve the use of the eyes. However, most of the senile macular degeneration patients can be assured that they will not become blind because the process remains confined to the central retinal area in a great majority of the cases.

There is no treatment known to be of value in the macular degenerations.

A major cause of decreased vision because of disease of the retina is retinal detachment. In the usual case a hole or tear occurs in the retina. Through this the vitreous fluid of the posterior portion of the eye makes its way and gradually separates the retina away from the underlying choroid. This process can proceed until the entire retina is loosened and extends forward in a sort of funnel from its attachment at the optic nerve. Any portion of retina which is elevated away from the deeper layers suffers degeneration, since it is out of contact with the blood supply derived from the choroid coat. The longer the retina remains detached the poorer the outlook, because of such degenerative changes.

There may be a definite history of a blow to the eye or the head which was sufficient to produce the original retinal hole or tear. In the majority, however, there is no such cause and the etiology must be assumed to be a degenerative change in the retina which produced a hole. This is likely to occur in highly near-sighted eyes where the retina is thinned out to line an eyeball which is longer than normal. In many normal eyes which are studied under the microscope there are found to be fluid-filled cysts in the retina at its forward limit just behind the ciliary region which are thought to give rise to many of the unexplained detachments. Quite obviously, if such degenerative changes are present, a retinal detachment may occur from a minor injury.

The patient with a beginning retinal detachment gives a fairly typical history. He notices first a shower of "soot-specks" before his vision. These probably result from the release of both blood cells and pigment granules into the vitreous at the time of the first retinal tear. Following this at varying intervals he will perceive a curtain gradually coming across his vision corresponding to the position of the progressing detachment.

As in many other eye conditions early diagnosis and treatment are most important in gaining a satisfactory result. Treatment of such cases is entirely surgical and consists of the use of diathermy current with small pins placed through the sclera in such a way as to form small spots of inflammation around the hole in the retina. The fluid beneath the detached area of retina is then aspirated off through the holes in the sclera and the patient placed in bed for 10-14 days in such a position that the retina may be sealed to the spots of surgical inflammation around the hole. Since the retina is not completely normal after successful reattachment it is most important that surgery be done before the macular area of the retina has come loose. If treatment is given after the central retinal area is detached the resultant vision is usually not better than 20/100.

Any patient who has had a detachment of the retina, or who appears to be a good candidate for one, must be warned against sudden blows or excessive straining, since these may aggravate the condition. Particularly in the cases which occur without any history of injury a retinal detachment is likely to occur eventually in both eyes, since the degenerative process leading to the condition is usually bilateral.

The results of surgical treatment vary with the length of time the detachment has been present and the extent of the area detached. Reports of as high as 75% success are found in the medical journals, but the average is probably closer to 50%.

There are a number of general diseases which produce damage to the retina resulting in loss of vision. In general such diseases act by interference with the blood supply to the retina due to damage to the vessels themselves. Diabetes is a major offender. Here are found hemorrhages in the retina which may be so severe as to stimulate the growth of scar tissue which proliferates into the vitreous cavity. The various types of high blood

pressure cause breakdown of the integrity of the small blood vessel walls, allowing blood cells and other blood constituents to escape into the retina, thus giving hemorrhages and patches of material which are known as exudates. Some of the blood diseases, such as pernicious anemia and leukemia, also result in the formation of hemorrhages and exudates in the retina, thus interfering with its function. The visual loss often corresponds in these conditions to the area of retina involved and if the central area is damaged central visual acuity fails.

Since the retina is supplied in great part by a single artery and drained by a single vein, damage to either of these vessels will cause severe damage. The central retinal artery enters the eyeball through the optic nerve and the central retinal vein leaves through the same region. A sudden closure of either of these main vessels produces a rapid loss of vision which is usually permanent. In the case of the artery it may be closed by an embolus (a solid particle of some sort), which is carried to the vessel by the blood stream and lodges in the artery. In other cases there may be a sudden narrowing or spasm of the artery. The patient notes sudden loss of vision and, if the process is unrelieved within 15-20 minutes, the loss is permanent. This is usually the case, since few instances occur that near to emergency care. The vein is more often blocked by pressure upon it from a neighboring artery which is diseased because of arteriosclerosis--a change associated with high blood pressure in many cases. The loss of vision is somewhat less rapid than in closure of the central retinal artery, but is often just as permanent.

In addition such a process may occur in any of the branches of the central retinal vessels in the retina itself. In such a case the area of retina involved ceases to function normally and a corresponding portion of the visual field is lost.

Tumors of the retina are just as much a danger to life as those arising from the choroid. They will be discussed later.

Vision

Before discussing vision directly it would be well to review the subject of light. Light is a form of radiant energy. The most common source in the daytime is the sun and at night an electric light bulb. Besides giving off light, both of these sources give off another form of radiant energy, heat. Light is very much like heat in many respects, the closer you are to the source, the more intense it is. Just as a light bulb when you come close to it feels warm and if you touch it it is hot, so, the closer to a source of light, the more intense or brighter the light. Also light travels in a straight line just as the radiant energy from the sun comes straight at you. If you get in the shade, you get behind something which stops the radiant energy, and it cannot curve around the tree leaves or building to get to you. Also, light can be absorbed just as the radiant energy of heat can be absorbed, as on a hot sidewalk in the sun. And, they can both be reflected just as an electric radiant heating unit has a reflector that reflects the heat in one direction, so light when it falls on an object is partly absorbed and part of it reflected. It is this reflected light coming from various objects which reaches the eye and enables the eye to see.

The light which falls on the sclera is absorbed by the pigment in the sclera and the choroid. The light which gets through the cornea then falls on the iris, is absorbed by the pigment in the iris and the pigment epitheleum. Any substance which acts in this way, that is absorbing the light rather than letting it get through, is said to be opaque. The only light used in seeing is that which comes through the cornea and aqueous, through the pupil, and then on through the lens and the vitreous. These are known

as the media of the eye. They are transparent, that is they let light through without distorting it. In contrast translucent material lets light through but breaks it all up so that it destroys the pattern, much as the threads on a weaver's loom might be messed up by a small child. This is seen in scarring of the cornea, where the light gets into the eye but is so distorted it is useless for vision.

Thus in seeing we have light coming from an object in a straight line to the eye and through the pupil. From here it goes back to the retina, but instead of going straight back and covering an area the size of the pupil, it is brought to a point. This is called refraction and is done by the optical system of the cornea and the lens. This is a complex procedure but it is easiest to think of the rays of light coming from a point and gradually spreading so that the whole pupil receives light from that point source, then this light is brought together again to a fine point on the retina. In the process, all rays of light can be thought of as crossing one point in back of the pupil. Thus objects that are down on the ground have their light come up through the pupil onto the upper portion of the retina. Objects which are high up have their light come down, into the pupil or down to a point in the lower part of the eye. Anything straight ahead has its light come right straight through the pupil to the back of the eye. This gives what is called an inverted image. The sensations produced on the retina are just reversed from what they really are on the outside.

The rays of light coming straight back fall on the area of the retina called the macula with the rest of the picture spread over the rest of the retina. In our description of the anatomy of the eye the macula was des-

scribed as having the highest visual acuity. The nerve endings here are all cones which are used in daylight vision for sharp discrimination, and the rest of the retinal layers are thinned out to give a sharper optical image. Thus, when the eye wants to see something very clearly it turns so that this portion of the retina receives the light from the object it wants to see. Away from the macula, the visual acuity or ability to make out differences in the light pattern, rapidly diminishes. This difference in sensitivity is much the same as the difference in sense of touch of the finger tips, the back of the hand and the back of the arm. An object looked at directly is seen with the macula and a very sharp clear image is received. But objects away from this one object are seen less and less distinctly.

Reports of eye examinations always carry the visual acuity. This visual acuity is a measurement of the ability to see with the macula. It is an arbitrary figure set down as a fraction with a numerator and a denominator. To find this vision, the examiner had the person look at a chart 20 feet away. This chart had rows of letters upon it. A large letter at the top, generally E, and lines of letters below this, each line smaller than the one above. The top big letter can be seen by the normal person and read at 200 feet. The letters just below it can be seen by the normal person and read at 100 feet, the letters below that at 70 feet and so on down, the next line 50 feet, the next line 40 feet, the next line 30 feet, the next line 25 feet, the next line 20 feet, the next line 15 feet, and the bottom line is generally the size read by the normal person at a distance of 13 feet. A person being examined is twenty feet from the chart and if he has normal vision he reads the line which should be read at 20 feet, giving him 20/20 vision, the top 20 being the distance from the chart, the

bottom 20 being the line a person with normal eyes would read at 20 feet. If he reads better than normal, that is the line a normal person would read at 15 feet, his vision is 20/15. However, if his vision is not as good as normal he will not be able to read the smaller letters and starting at the top he will only read part way down, say to the line a normal person would read at 70 feet. Then his vision is recorded as 20/70 or if he could only see the top letter, then his vision is recorded as 20/200, that is, at 20 feet he could only see the size a letter a normal person could see at 200 feet. If he cannot see the top letter at twenty feet, the examiner has him move gradually forward until he can make out the top letter, and then his vision is recorded with the distance where he can see it over 200. If he can see it at 10 feet his vision is 10/200. If he sees it at five feet, his vision is 5/200. If the person cannot see the top letter at all, then the examiner holds up his hands to see if he can count fingers. If he can discriminate how many fingers the examiner is holding up, the examiner gradually moves away to see at what distance he can still count fingers, and this is recorded as "count fingers" and then the number of feet away at which he can count fingers, counts fingers two feet or counts fingers four feet. If the person cannot count fingers, the examiner takes two small lights and holds them up about three feet from the person, and gradually moves them apart, turning them on and off, sometimes one and sometimes two, seeing at what distance they have to be separated before the person can tell how many lights there are. Also turning the light off and on he moves it up and down and to either side, seeing if the person can tell which way the light is coming from. This is called light projection. The examiner also turns on different colored lights to see if he can recognize the colors.

Then finally, if all of these seem defective, he tests to see whether the person can tell whether a light is on or off, light perception. All of these tests, except light projection and perception, are tests of the ability of the macula. The last tests are used when the lens or cornea are still translucent but are so changed that light cannot get through without marked distortion, and thus the examiner cannot see into the eye to tell what condition the retina is in.

"Visual acuity" shows the function of the macula. To test the rest of the retina an examination is made of the "visual fields." Here the person examined is seated before a large blackboard, one eye is covered, and he looks straight at a white dot in the center of the board. This fixes his eye straight ahead at the one spot. He looks at this spot with his macula. Now, to test how well the rest of the retina is functioning, the examiner takes a small round disc or ball on the end of a black wand and starts way out on the edge of the board, moving it back and forth and gradually bringing it in toward the center. The person being examined tells the examiner as soon as he sees this test object, or white dot. The examiner generally starts from one side and as soon as the person sees the object, the examiner makes a mark on the board. Then he starts in a little above this and comes in again. Gradually he works all the way around the board, marking as soon as the person being examined says he can see the object. After this, a larger test object is used, being brought in from the sides in the same fashion. Being larger, it is easier to see and is generally seen further out. In this way the function of the whole retina can be tested, and marked out on the board. This in turn is copied down onto a piece of paper, giving a permanent record of the function of the retina at that time. Different

diseases will give different visual fields. Thus, a study of the visual fields shows how much peripheral vision the patient has and also helps as a diagnostic procedure. When there is an injury to the retina as in a chorioretinitis, the area involved is blind while the area around it can still see, and these areas can be mapped out quite accurately on the visual field. In glaucoma a very characteristic visual field is obtained. As the pressure, which will be described later, is applied on the optic nerve at the nerve head, the nerve fibers going into the optic nerve are injured. In a glaucoma simplex this is a gradual process and the loss of vision is not noticed as it is a peripheral loss. But with the visual fields, a definite pattern is found on examining the patient. With lesions of the optic nerve, as in a retrobulbar neuritis, the different forms of inflammation cause different types of defects in the field, and injuries to the visual pathways on back give a definite type of pattern for each area which is involved, thus helping the doctor to localize the point of injury.

Thus, in checking vision, first the central vision is tested to find how well the macula is functioning. Then, if indicated, visual fields are taken to see how the rest of the retina is functioning. Both of these are important. A person may have very poor central vision, down to 20/200 or less, and still be able to get around very well and walk through downtown traffic because the rest of his retina is functioning perfectly and he can stay oriented to his environment. On the other hand, a person may have 20/20 vision when looking straight ahead at the vision chart. However, he has to be led around because all of the rest of the retina has been destroyed except a small area at the macula.

Just what constitutes blindness has been difficult to decide. For practical purposes, it is sufficient loss of vision to prevent the person from pursuing his usual occupation of earning a living, in contrast to absolute blindness where even light perception is lost. When central vision in the better eye is 20/200 or worse it is considered economic blindness. When vision is better than this it is useful vision with a percentage loss. The exact percentage is figured by using tables which consider both the central visual acuity lost and the amount of visual field lost.

Refraction and Muscles

Refraction is a study of how light is bent as it passes through the eye. Our main interest is in how glasses help some people to see better.

In the last chapter it was described how light coming into the eye from a point source was again brought to a fine point upon the retina of the eye. An eye is often compared to a camera, as it has a similar arrangement. The light comes to a camera through a shutter like the pupil, on through a lens like the lens system of the eye, then back to the film in the camera which can be compared to the retina of the eye. For focusing a camera there is a bellows arrangement which allows the lens to be brought closer to the film of the camera or further away. This is necessary because when an object is brought closer to a camera the rays of light are brought to a point focus further behind the lens. So, to get the object in a clear focus the lens has to be moved forward away from the film of the camera. Otherwise it would be out of focus. In the same fashion when the object is brought further away from the camera, the rays of light are brought to a sharp point focus closer to the lens, thus the lens has to be moved back toward the film in the camera. On the other hand, the shape of the eye is fixed. If when looking in a distance the rays of light are brought to a sharp focus on the retina, it is a normal eye. However, if the eyeball is too long, in looking at a distance the rays of light are brought to a point focus too close to the lens, in front of the retina. However, as the object is brought closer to the eye, this point focus of the light moves back toward the retina until the eye is in focus. This is a near sighted eye. Things nearby are in focus, but further away, because the eyeball is too long, the object is out of focus. The eye has no way of overcoming this handicap, as it cannot shorten itself and has no muscle to change the lens to move its point of focus back.

On the other hand, if the eyeball is too short, the rays of light if their path could be extended through the retina would come to their point focus behind the retina. This, in contrast to the near-sighted eye, is a far sighted eye. Here the eye can do something about it. The ring muscle and the ciliary muscle can constrict down, relaxing the zonular fibres which in turn relax their hold on the lens allowing it to become more round. And, then, when the lens is more round it brings light to a focus closer to the lens and hence the eye is in focus. So a near sighted eye is a long eye, and can do nothing to bring objects into focus except to bring the object itself closer to the eye. On the other hand, a far sighted eye is too short, and by working, it can overcome its handicap.

The third type of refractive error is called astigmatism. Here, the front part of the eye, the cornea, instead of having a regular curve like a ball or the end of an egg, has a sharper curve one way than the other, like the side of an egg, where the short axis has a sharper curve than the long axis. With a cornea of this type, causing astigmatism, light from the point source cannot be brought to a point focus for it is bent more in one meridian than in the other. Because of this the light is brought to a line focus. To correct these errors, the doctor examines the eyes and finds out exactly what the error is, then prescribes glasses which compensate for the error to make the eye artificially normal. Thus, in a near-sighted eye, a minus lens is prescribed which moves the point of focus back to the retina. In a far sighted eye a plus lens is prescribed which brings the point of light forward to the retina. And in astigmatism a lens is prescribed which has a stronger curve one way than the other. This counteracts the difference in curvature of the front of the eye, so that light is brought to a

point focus on the retina. Thus, glasses help an eye to see better by helping to bring rays of light to a focus on the retina. Glasses are of no help to an eye which has any other difficulty. Glasses will not help light to get through a scarred cornea or an opaque, cataractous lens. Glasses cannot help a diseased retina to see better. They serve the one purpose of bringing light to a sharp focus on the retina when the media of the eye are clear.

When an individual looks at an object, he not only focuses each eye clearly, but each eye must be pointed at the same object. This is done for two reasons. One, to get the eye pointed at the object so an image one wants to see falls on the macula. The second reason is to get the same image falling on the same part of each eye. Otherwise, the individual will see two pictures of the same thing, one with each eye. This is called diplopia. Ordinarily the two eyes move together like a couple waltzing, up and down together, side to side together. This allows the brain to "fuse" the two pictures into one. When there is a paralysis of one of the extra-ocular eye muscles, that eye has difficulty keeping up with the other, causing what is known as a diplopia. In diplopia, the individual sees two images just alike instead of one. Because a person with normal vision depends so completely on vision to orient himself to his environment, this diplopia is most disconcerting. A mild idea of diplopia is obtained when two network stations carrying the same program are too close to one another on the dial and the program on one station for some reason comes in a split second behind the other with both of them equally loud and imperative of your attention.

When this occurs in an adult it can sometimes be corrected by the use of a prism. A prism bends light so that it can be bent to line up with the

eye. Sometimes this does not work and it is necessary to blindfold one eye, thus eliminating one image. When the condition becomes quiet and shows no signs of improvement it can sometimes be helped by operating on one of the extra-ocular muscles to line up the eyes properly. In children, when one of the eyes crosses, the resulting diplopia is just as bothersome to the child, but he soon learns to suppress that eye. That is, he just doesn't see the image that comes in that eye, just as when you put your shoes on in the morning you forget about them and don't think about them again until you read this bit. This is all right as a temporary measure, but the child continues not to use the eye; then, just like carrying an arm in a sling for a long time, it gets so that it is of no use. This eye which is not used for seeing gets so that it cannot see well, and for practical purposes the child has a blind eye. For this reason, all children with crossed eyes should be seen by the ophthalmologist as soon as possible. With proper treatment the vision in the eye can generally be preserved, if the child is seen early enough.

Diseases of the Eyelids, Conjunctiva, and Cornea

The eyelids act as the protectors of the eye. Any diseased condition of the eyelids will cause discomfort in themselves, and also if they disturb the function of the eyelid, will cause damage to the eye proper. The congenital anomalies of the eyelids include such unusual conditions as no lids being present, called ablepharon, adhesions between the two lids, complete or partial, called ankyloblepharon, and defects in the eyelid itself, called coloboma. These are all of varying amounts and if severe require surgical procedures to correct them. The most common congenital defect is a drooping of the upper lid. This is called a ptosis. Here the upper lid cannot be elevated, so that it hangs in front of the eye and prevents the light from entering through the pupil and back to the retina, when the head is held in the normal position. To overcome this the person must tilt his head way back so that he can peek out from under his eyelids. To remedy this, various surgical procedures are employed.

The skin of the lids can become involved in any inflammation of the skin involving the rest of the body. Also, because the skin is so thin here, it is more frequently involved in allergic reactions. Sometimes it is an allergy to drugs and often it is an allergy to cosmetics such as the face powders, fingernail polish, etc. At times this is a handicap to a doctor in treating an eye condition, because the skin of the lids becomes sensitive to the medicines used, such as stropine, and swell up markedly. The lid margins will frequently become involved in a chronic inflammatory condition which does not bother the vision but which is quite irritating to the patient. If not treated it gradually causes a thickening of the lids and is very difficult to clear up. A common form of inflammation of the lid margin is a stye, or hordeolum. This is like a boil. It is an acute inflammation of one of

the glands at the base of an eyelash, generally caused by a staphylococcus. This causes considerable local swelling and redness and considerable pain. It is treated with applications of heat and some medicine such as sulfathiozole. Then, when it becomes localized to a point directly under the skin it is incised with a sharp knife. The meibomian glands which extend deep into the lids can also become involved in a similar acute inflammation. This is generally more severe and has more swelling of the lids, but is treated in much the same way. These glands also will sometimes become blocked. The secreted material which accumulates causes the gland to swell making a "bump" in the eyelid which sometimes will get up to the size of a large pea, or larger. The only treatment for these is to open them up surgically and remove the contents.

A Herpes Zoster, like shingles on the chest, can affect the ophthalmic division of the fifth nerve. This will cover the forehead and the upper lid and nose. The area becomes reddened, very painful and covered with small vesicles. If the condition is limited to the skin, it is not of too serious an import, but it can also involve the cornea and the iris. Here, if the scarring is too bad there may be permanent loss of vision in the eye.

The lids may have a change in their position, being turned out, ectropion, or turned in, entropion. Ectropion can come from a variety of causes such as extensive scar formation following an injury, a burn, or inflammation of the skin, or it may follow senile atrophy of the skin, pulling the lid down. With the lid pulled away from the eye the tears cannot get into the lower puncta, so they flow out onto the skin of the lower lid causing an irritation and more shrinking of the skin with more ectropion. This condition is irritating because the eyeball and palpebral conjunctiva are ex-

posed, causing itching, burning and increased lacrimation. If it is severe, the cornea is exposed and in time, with the loss of protection from the lower lid, the lower cornea may ulcerate. When the condition is at all severe it requires some surgical procedure to put the lid back in its proper place.

Entropion or turning in of the lid may be caused by scar tissue or a spasm of the muscles of the eyelid. When the lid is turned in, the lashes of the eyelid rub against the cornea. The cornea being very sensitive, this irritation causes the patient to blink his eye. This in turn again rubs the lashes on the cornea and sets up a vicious cycle of irritation and spastic closing of the eyes. The treatment here too is surgical repair of the defects.

Tumors of the eyelids are of different types. Some are perfectly benign. These the doctor leaves alone unless the patient desires their removal for cosmetic reasons. Carcinoma of the skin is frequently seen on the eyelids. Fortunately when it appears here it is in a conspicuous location and if seen early, it can be removed surgically in its entirety. If not seen early, of course, it will continue to grow and invade requiring extensive surgery and radiation for its treatment.

Injuries to the eyelids can be like injuries anywhere else, and of any extent. The most commonly known is the black eye. This is like a bruise anywhere else in the body, but because of the looseness of the tissue under the skin, considerable amount of blood escapes into it, ballooning it up and making the eye very conspicuous. In treatment ice is applied early to cut down on the amount of bleeding and later on heat helps to speed up the absorption of the blood. Wounds of the eyelid are treated surgically and they require very exact care in the closing up of the cut tissues. If the tissues

are not put in direct apposition, layer by layer, the resulting scar tissue will draw the lid into distorted forms preventing its serving as protection for the eye. If the distortion is at all extensive, plastic surgery should be done at a later date.

The conjunctiva, or lining of the posterior surface of the lid and the front of the eyeball, can become infected by a number of organisms, causing a conjunctivitis. Different cases of conjunctivitis caused by different bacteria often will present a very similar picture to the doctor when he examines the eye. Because of this, if the inflammatory picture is at all severe, the doctor will make cultures from the conjunctival sac and also gently scrape along the lining of the lid to get smears to be examined directly under the microscope. This way, by knowing the exact organism which is causing the inflammation, he is better able to treat the condition. Probably the most common inflammation of the conjunctiva is the acute catarrhal conjunctivitis, or pink eye. This can be caused by a number of organisms all presenting the common picture of an acutely inflamed conjunctiva. There is very little pain with this, but there is a foreign body sensation, a mild photophobia, which is discomfort in bright light, and some disturbance of vision by the secretions. Unless it is very severe the cornea itself is not involved. The condition is quite self limited and with the newer drugs clears up quite rapidly. However, it is a definite nuisance, especially as it is highly contagious and will rapidly spread through a group such as a family or a barracks of soldiers. Chronic catarrhal conjunctivitis includes any chronic inflammation of the conjunctiva and again can be caused by a variety of organisms, also by chemical irritants such as smoke and dust and occasionally by an allergy to certain substances. The clinical picture is a

mild injection of the conjunctiva with mild irritation of the eye. If an allergy causes it the patient frequently complains of itching. The diagnosis is difficult at times and again requires cultures and scrapings and a careful history. The treatment is directed towards the organism or the allergy causing the condition.

Besides a catarrhal type of conjunctivitis an allergy can cause a seasonal type of inflammation of the conjunctiva called vernal conjunctivitis. Here there is a thickening of the conjunctiva, either of the conjunctiva on the lids or of the bulbar conjunctiva next to the cornea. The main symptom is itching which may be quite aggravating. It is often very difficult to find the exact substance to which the person is allergic, so that treatment has to be symptomatic. Sometimes weak solutions of epinephrin are used, sometimes just a normal saline or sodabicarbonate solution to keep the conjunctival sac clean. In some patients the recent antihistaminic drugs, such as benadryl, which are used for hay fever give a great amount of relief.

At times the inflammation of the conjunctiva may be so severe it causes a pouring out of secretion and serum which coagulate over the conjunctiva forming a membrane. All of the organisms which cause a catarrhal type of conjunctivitis can cause this type of membrane to form. This is called a pseudo-membranous conjunctivitis, in contrast to a true membranous conjunctivitis where the organism causing the conjunctivitis is the true diphtheria bacillus. In membranous conjunctivitis the diphtheria is the same as if it were in the throat except the organism is growing in the conjunctiva rather than in the membrane of the throat. If not treated it can go on through the same picture of any diphtheria case. The other forms and the diphtheria form

if severe enough can involve the cornea causing permanent scarring or even perforation with infection of the eyeball itself.

Trachoma is a familiar name to all who work with the blind. In this country it is relatively rare being found only in certain areas, as among the Indians, in Tennessee, Kentucky, and Ohio. It is also found in southern Illinois, South Dakota, and Nebraska. However, it is becoming less and less frequent. The cases now being seen by the ophthalmologists are generally old cases that have been present for years rather than new acute cases. In other parts of the world such as Egypt and Palestine and China, as high as 90 per cent of the native population is affected. It is caused by a filtrable virus. The clinical picture is generally divided into four stages, the period of onset, the hypertrophic stage, the cicatricial stage and the healed stage. The period of onset presents only a mild catarrhal type of conjunctivitis with thickening of the conjunctiva. But this continues and small bumps called follicles appear on the upper and lower tarsal conjunctiva. These gradually continue to grow for three or four weeks. During this time there are very few symptoms. Gradually it progresses into the second, or hypertrophic stage. At this time the symptoms generally become severe enough to bring the patient to the ophthalmologist. The follicles are larger now so that they give a very velvety appearance to the conjunctiva. The lids now seem thickened and are a beefy red. Also at the upper part of the cornea, vessels can be seen growing outward over the cornea, beginning what is known as a trachomatous pannus. This pannus may be very slight or it may extend down over the pupil causing permanent scarring with loss of vision. Gradually the acute symptoms subside and the disease passes into stage three, the cicatricial stage. Here the eye is healing up but as it heals the tissues

contract down because of scar tissue which forms. This scar tissue as it contracts distorts the lid if it is at all extensive and appears as very fine network of white lines criss-crossing on the lid. This is a gradual process and may go on for years, constantly contracting and distorting the lids. Finally the stage four, or healed stage is reached where the disease is an arrested condition. If the course is mild there will be a small amount of scarring. With increased severity the lids are more distorted and the pannus more extensive and it may go on to complete distortion of the lids causing an entropion of both lids and a pannus over the cornea so that light cannot get through without complete distortion. The treatment of this condition up to recent date has been the use of copper in some form, generally in the form of crystals of copper sulphate which are applied to the conjunctiva after the installation of a local anesthetic. This is done every day. This procedure is quite painful at first although in time the patients get so that they don't mind it too much. Recently sulfathiozole has been used both locally in the eye and generally, taking it by mouth, with very excellent results, so that as a rule extensive damage to the eyes is not seen today like it used to be. In the stages three and four, if the scarring is extensive, surgical procedures are sometimes necessary.

Another condition whose appearance has changed radically in the last few years is the gonococcal conjunctivitis. Gonorrheal ophthalmia neonatorum, or the gonorrheal infection of the new-born used to be a dreaded condition. It is an inflammation which develops between 24 to 48 hours after birth in the eyes of the new-born with marked swelling of the lids, an extreme amount of pustular discharge and the frequent complication of corneal ulceration with perforation of the cornea. Formerly the prime treatment was almost con-

stant irrigation of the eyes, to wash out the irritating pustular secretions and thus protect the cornea. The large hospitals in the cities often had a nurse who specialized in just this one condition and would stay constantly with the patient. The Crede Law, requiring a drop of one or two per cent silver nitrate in each eye of a new-born child, cut down markedly on the incidence of this condition. Now, with the advent of penicillin, treatment is instituted as soon as the condition is diagnosed and it clears up very rapidly.

The gonococcal conjunctivitis in the adult generally occurred by accidental self-inoculation from a genital infection. Here the picture was of a similar type of that seen in infants but more severe. It could generally be kept to the one eye. Corneal complications were much more common. Today with the advent of penicillin the condition responds rapidly to the proper treatment.

Pemphigus of the conjunctiva occurs with the Pemphigus over the skin of the body. Pemphigus is a condition of the skin where large blisters form and rupture leaving a raw exposed surface which heals with scarring. It has a devastating effect upon the eye, for, as the blisters form and rupture and heal with scarring, there is marked distortion of the lids. The conjunctival folds become obliterated by scar tissue and the cornea, because of the exposure and the turning in of the lashes, or trichiasis, becomes irritated, vascularized and opaque. Here the treatment is disappointing. Membrane graft from the lining of the lips has been tried without success, and surgery for the scarring of the lids generally does not do a great deal of good.

New growths of the conjunctiva are quite rare except for one benign type called a pterygium. Here the bulbar conjunctiva in its exposed portion

becomes irritated and develops many vessels. Then at the limbus these vessels begin to migrate over the cornea. If they do not grow far they cause no damage. However, if they extend over the pupil, vision is lost. The treatment here is a surgical procedure to lift up this new growth off the cornea and then point the tip of it down under the conjunctiva of the eyeball. If the growth is just cut off, it is very apt to recur. However, by lifting it up at its tip and then burying the tip under the conjunctiva below the cornea, it continues to grow in a harmless position.

Injuries to the conjunctiva whether they are chemical burns or mechanical injuries are like injuries to the lid in that if they are extensive they will cause distortion of the conjunctiva and of the lid, requiring surgical correction.

The Cornea

The cornea forms a protective coat on the front of the eye just as a crystal in a watch protects the watch. It has no blood supply of its own, obtaining its nourishment from the aqueous in back of it, the tears in front of it, and from the blood vessels around the limbus. When there is any injury or disease to the cornea it is rather slow to heal because of its lack of blood supply. Certain conditions do not heal until the blood vessels grow into the cornea from the limbus to the involved area, thus providing sufficient nourishment. The cornea in its natural state is perfectly transparent, allowing light to pass through without distortion. When it heals, following an injury or disease, the resulting scar tissue is not laid down in the fine regular pattern of the normal cornea, but in a more irregular dense fashion. This, of course, distorts the light as it passes through. For this reason, any involvement of the central portion of the cornea is very serious. It can heal up completely, but it heals up with a scar tissue which does not allow light to pass through properly and thus vision is destroyed, although the rest of the eye functions normally.

Inflammatory conditions of the cornea are generally divided into those which are on the surface of the cornea and those which are deep. An inflammation of the cornea is also called a keratitis.

Superficial inflammations of the cornea or superficial keratitis causes ulcerations of various types. The severe acute ulcerative keratitis is called a serpent ulcer, a hypopyon ulcer, or serpiginous keratitis. Like an acute conjunctivitis this condition can be caused by a variety of organisms, most frequently a strepticoccus, or a neumococcus. The organisms generally get in through a break in the epitheleum made by a minor injury or by a foreign body lodging on the front of the eye. Early there is gray in-

filtration around the site of the injury or foreign body which gradually spreads. As it spreads the eye becomes painful and acutely inflamed. The ulcer spreads just under the epithelium undermining the epithelium as it grows, and it generally heals at one edge as it progresses on the other side.

The toxins of the infection not only cause marked injection of the bulbar conjunctiva but also set up an inflammatory process in the iris. This causes a pouring out of pus cells into the anterior chamber which can be seen through the cornea, a hypopyon. If the ulcer continues to progress it may work deep into the corneal stroma and perforate into the anterior chamber of the eye. Then the aqueous of the eye escapes collapsing the anterior chamber. Some times the iris, and even the lens, is washed forward into the perforation; and when it heals, they heal in this position. The infection may pass on into the interior of the eye setting up an infection throughout the eye, or panophthalmitis. The process may heal up at any time in this picture. If it heals early just a small scar is left. If the scar is away from the pupil, no permanent damage is done. If the scar is over the pupil, useful vision is of course cut down or even destroyed. Very often, following a perforation the ulcer heals spontaneously, and if the perforation is away from the center of the pupil some useful vision may be retained. Because of the pus and irritation in the anterior chamber, and also from the iris being pulled forward into a perforation, these cases often result in the blocking of the chamber angle, with a resultant rise in intraocular pressure, causing a secondary glaucoma. The corneal ulcers are treated with antibiotics. Sometimes it is necessary to cauterize them and very infrequently it becomes necessary to make a delimiting caratotomy. In this the surgeon makes an incision through the cornea just in front of the advancing edge of the ulcer. This generally stops the progress of the ulcer.

A catarrhal or marginal ulcer occurs just inside the limbus superficially and runs a much milder course. Here again a variety of organisms can cause the condition. There is generally a chronic conjunctivitis associated with it. The symptoms are much milder than in the serpiginous ulcers. Treatment is directed toward the chronic conjunctivitis and at times it is necessary to cauterize the ulcer itself.

Rarely an ulcer will start like a catarrhal ulcer but instead of responding readily to treatment, it will gradually continue to spread at both ends and toward the middle until it will involve the entire cornea. This is called a chronic serpiginous ulcer, or Mooren's ulcer. Treatment of these cases is difficult. Sometimes one will respond if the surgeon covers it completely with a flap of conjunctiva.

The herpes virus will at times attack the cornea causing a Dendritic ulcer. This starts as a row of very fine blisters which break and coalesce causing a fine line on the cornea. Branching rows of blisters will sometimes follow, which then break and form lines so that it has the appearance of the limbs of a tree. The eye is quite painful and there is considerable injection. The response to treatment is quite variable, some clearing up promptly with local applications of iodine, others taking considerable time and care.

During the war a condition known as epidemic kerato-conjunctivitis, or shipyard conjunctivitis, was frequently seen in epidemic form along both the east and west coasts and along the Mississippi valley. Here there was an acute conjunctivitis accompanied by a large number of superficial small lesions of the cornea. No known treatment seemed to have any great effect on the course of the disease other than to make the patient more comfortable.

The cornea depends upon the lid for protection and indirectly for nourishment as it brings the tears over the eyes. When the lid fails to cover the eye a condition known as exposure keratitis develops. This is generally caused by paralysis of the facial nerve controlling the orbicularis oculi muscle closing the lid. It can also occur when there is an extreme exophthalmos in hyperthyroidism or with an orbital tumor causing the eye to bulge forward, or following loss of the eyelids, or when a person is in coma for some time lying with his eyes open. The exposed portion of the cornea loses its normal shining luster and becomes dull. Then it gradually ulcerates presenting the picture of a corneal ulcer. The treatment is directed first at the secondary infection with some antibiotic and then to keeping the eyelid closed so as to give normal protection to the cornea. When this is done, the cornea heals quite rapidly.

Destruction of the trigeminal branches to the cornea, those which carry sensations from the cornea, results in a condition called keratitis neuroparalytica. Here the eye loses its normal protective mechanism of winking. With any irritation of the eye which would normally cause a winking and brushing away of the irritation, the eye now does not feel it. It stays open, and leaves the dust accumulate, or whatever is sticking it go ahead and injure the cornea undisturbed. These nerve branches are generally destroyed intentionally in a surgical procedure, so the condition can be watched for, and at the first signs airtight goggles are given to the patient. Also, he is advised to watch the eye at frequent intervals to see if any dirt has accumulated or if there is any sign of irritation, injection of the conjunctiva. He is shown how to clean out the conjunctival sac each day.

Deep inflammations of the cornea include two important forms, inter-

stitial keratitis and disciform keratitis. Interstitial keratitis is a result of congenital syphilis, or rarely tuberculosis. The Wasserman, a blood test for syphilis, is generally positive and other signs of congenital syphilis are present such as deafness, characteristic notching deformities of the teeth, skin lesions about the mouth and a flattening of the bridge of the nose. It generally starts between the fifth and fifteenth years of age, most frequently around ten to twelve. The cornea develops a grayish opacification of the middle layers of the stroma with edema of both the epitheleum and the endotheleum. The vessels of the conjunctiva around soon become injected and later vessels start growing into the stroma of the cornea. They grow in between the layers of the stroma at all levels. On cross examination they give an orange red appearance to the area where they are growing in, gaining the name of "salmon patches." There is often an accompanying iritis. Both eyes are generally affected although one can come on quite a bit later than the other. The first symptom is irritation of the eye, which develops to the point of severe photophobia. The child often will lie with his head buried in a pillow and refuse to allow an examination of his eyes until medicine has been put in to cut down the pain. There is marked diminution of vision because of the opacities. This will go on for a six to eight weeks period. Treatment has little effect on the course of the disease once it has started. The results can be anything from normal vision to less than 20/200.

Disciform keratitis occurs in adults. It presents a picture of a gray infiltration in the stroma of the cornea. There is generally only a small amount of congestion of the conjunctiva. Occasionally there may be moderate irritation with some pain and lacrimation. This is a chronic condition which will go on for several months, and gradually begin to clear. Here again the

results are variable from almost normal vision up to loss of all useful vision. The treatment is of questionable value here also, although heat and fever therapy are generally employed.

The cornea can be involved in a variety of degenerative processes. The most common is called the arcus senilis. Here there is a gray infiltration of the cornea just inside the limbus. Although it is sometimes very conspicuous it very rarely advances to the point where it cuts down on the vision. The epitheleum at times in people past 45 will develop small blister-like formations which can sometimes be seen only with the slit lamp. This is called Fuchs epitheleal dystrophy. This will sometimes reduce the vision a moderate amount. The endotheleum also has a form of dystrophy in which little hyalin nodules are formed just underneath the endotheleum in Descemet's membrane. These, too, will sometimes reduce the vision, as low as 20/70. In neither of these is any treatment of any avail. There are other more rare forms of dystrophies which will at times run in families in an inherited pattern.

In chronically diseased eyes, such as ones with glaucoma, when the vision is lost for some time, the cornea will sometimes develop an opacification of the exposed portion called "Band shaped" opacity. This consists of calcium salts deposited in Bowman's membrane, at times to such an extent that it can be lifted off in its entirety.

Keratoconus is the name of a condition in which the curve of the cornea changes. The central portion of the corneal stroma becomes thinner, allowing the cornea to bulge at this point. The cornea instead of being "dome-shaped" becomes more "cone-shaped." Hence the name. This condition is

gradually progressive, and there is no treatment which seems to help. Because of the optical defect it is sometimes difficult to prescribe glasses which do much good for the patient. In some of the more advanced cases contact glasses are of great assistance, making the difference between useful and non-useful vision.

Sympathetic Ophthalmitis

Sympathetic ophthalmitis is a rare form of uvietis, or inflammation of the vascular coat of the eye. The typical story starts with an injury to the eyeball which penetrates, injuring the ciliary body. The injury then is slow to heal with the eye remaining in an irritable condition. Then, gradually a very slow insidious inflammation sets up in the other eye which can continue to total blindness in the uninjured eye.

On microscopic examination the picture is somewhat similar to that seen in tuberculosis. It has the same type of chronic inflammatory cells, but with an arrangement which is characteristic of sympathetic ophthalmitis. Although this condition has been known and dreaded for a long time the cause has never been found. A vast amount of research and speculation has been expended but nothing has been definitely found. Some have felt that a special type of organism traveled up the optic nerve of the injured eye and then down the optic nerve of the other eye. Others have felt that the patient became allergic to his own uveal pigment, but nothing has ever been proven.

There is always a penetrating injury involving the ciliary body which heals slowly. It is rarely found to follow a wound that heals clean and also rarely known to follow a wound to an eye which becomes infected developing a panopthalmitis.

The inflammation of the uninjured or sympathizing eye generally begins four to eight weeks after the injury. This inflammation develops very gradually. The first thing noticed is a gradual loss of vision with very little discomfort or injection. With slit lamp examination a few cells can be found in the anterior chamber and some fine deposits on the back of the cornea. Then the eye becomes more irritable, as to a normal examination. Then gradually injection appears around the cornea. The inflammation be-

comes more acute and may continue on to complete destruction of the eye.

The obvious treatment here is to remove the cause, the injured eye. This decision taxes the judgment of the attending ophthalmologist. An injured eye which is so mutilated that no vision remains and a prosthesis would present a better cosmetic result, should be removed. An injured eye which is slow in healing and irritable with vision down to poor light projection should be removed. However, when the eye appears as if it might retain useful vision the decision is difficult. Here the ophthalmologist generally makes every effort to save the eye at the same time closely following its course, and also keeping close tab on the uninjured eye. When a patient comes for treatment with this condition already developed in the uninjured eye it is generally best to leave the injured eye for in some cases the final results show a better vision in the injured eye than in the uninjured eye. Also, once this type of inflammation gets well started in the uninjured eye, its severity does not seem to be limited by removing the injured eye. The treatment here is that of any uvietis, including atropinization, foreign protein therapy, etc.

The Eye in General Disease

Cardiovascular-Renal Disease

Diseases of the heart, the blood vessels and the kidney occur together so often that they were soon found to be closely interwoven. The three main factors here include hypertension, or high blood pressure, arteriosclerosis, or hardening of the arteries, and renal disease or kidney disease. These can occur independently but as a rule when one exists for a while, the others are then found, and when the picture is seen in an advanced state it is difficult to tell just what started first in the patient. Sometimes one predominates and sometimes another.

Arteriosclerosis

Arteriosclerosis is a degenerative process involving the walls of the arteries. It may be of a patchy type or it can be a diffuse process. The vessel walls have a fatty substance deposited in them which in time may become calcified. In the larger vessels this can convert the vessels into regular pipes, but still it does not occlude the vessel lumen. In the smaller vessels the process is generally diffuse with a thickening of the vessel wall. However, in the small vessels, as the walls become thicker there is not much room for expansion and the lumen, or the hole in the artery, is made smaller and smaller. In time it may even close. There is no known cause for this process. It generally comes on in older people.

The vessels in the eye grounds can be looked at directly by the ophthalmologist, and when they show signs of sclerosis he can tell it. The arteries normally are transparent, showing just a column of blood flowing through them. But, as the walls become sclerosed, the walls become visible, and where the artery crosses the veins the veins cannot be seen. Also, if

the artery wall becomes more sclerosed, the vein is pushed to one side, and if the process continues, it may even block an artery, or at a crossing a vein can be occluded. In this way an occlusion of either the central retinal artery or vein can occur with complete blindness ensuing, or, less spectacularly, one of the small branches can be blocked causing exodates and hemorrhage in this area.

The same process that is seen in the eye also takes place over the body, most frequently in the heart and in the kidneys. The blocking of the small arteries going to the kidneys causes a starvation so to speak of the area of the kidney supplied, hence that area cannot function. The same thing happens in the heart where the coronary arteries, the arteries coming off of the aorta and going to supply blood to the heart itself, also become blocked off.

The areas of fatty infiltration in arteriosclerosis are weak points in the vessel wall. In the brain these areas will rupture allowing blood to escape into the brain tissue. That is what happens commonly with a stroke. As the blood is absorbed, some function is regained, but many of these people will have "miniature strokes." Tiny vessels are occluded and have ruptures causing minute hemorrhages into the brain. As these occur again and again the brain gradually loses its efficiency. This is the reason for many of the senile changes in personality.

As the normal heart pumps the blood out into the blood vessels, it puts the blood under a pressure to keep it flowing. In hypertension, the blood in the blood vessels is under greater pressure. This comes either from the heart pumping harder or from increased resistance in the smaller vessels, the arterials. The cause of hypertension is not known. It apparent-

ly occurs as a compensatory mechanism to keep the body functioning normally. At times something apparently goes wrong with this compensatory mechanism, and the blood pressure remains elevated. Maintaining this elevated pressure puts a greater load on the heart so that it has less reserve and is more subject to stress and also it puts a greater load on the blood vessel walls so that they are more prone to rupture allowing blood to escape, as is especially seen in the brain. And, as time goes on the walls are inclined to become thickened, arteriosclerosis.

The Ophthalmologist on examining the blood vessels of a patient with a hypertension, finds the changes which were described in detail in the chapter on retinal diseases. These are basically a spasm of the artery, either a generalized spasm or a generalized attenuation, or a local spasm. When the disease has progressed further the other changes follow of hemorrhages and exodates, arteriosclerosis, or papilledema.

A diseased kidney can in some way cause an increase in the blood pressure. This increase in the blood pressure in turn makes it more difficult for the kidney to function and in time will cause a sclerosis of its vessels. This in turn, causes more damage to the kidney. As the kidney is more damaged it is more prone to cause a further rise in the blood pressure and a vicious circle is set up. Hence the term cardiovascular-renal disease.

The fundus picture seen in arteriosclerosis and in hypertension, and in nephritis have been described in the chapter on diseases of the retina. Although they each have a more or less characteristic picture they all stem from changes in the vessel wall, allowing an escape of serum first and if more severe, allowing an escape of blood, making small hemorrhages into the retina.

Metabolic Diseases

In the section on anatomy two nervous systems were described. One the voluntary nervous system which directed activities at the conscious level, and the other, the autonomic system, which directed activities below the conscious level such as breathing, functioning of the heart and the intestinal tract. The body chemistry is also controlled below the conscious level. This control system is composed of the glands of "internal secretion." We are well aware of how the lacrimal glands secrete tears, and the salivary glands secrete saliva. The body also has glands which secrete their material into the blood stream. This material in turn controls different body chemical reactions by stimulating or inhibiting them. These glands are controlled both by the autonomic nervous system and by the chemical needs of the body. The adrenal glands are an example of glands under the control of the sympathetic nervous system, for under excitement they pour their secretions into the blood. These secretions stimulate the body in its preparation for defense or offense. On the other hand the pancreas has cells in it which occur in little nests of cells called the Islets of Langerhans. These secrete a substance called insulin. This substance is secreted into the blood stream whenever the sugar content of the blood stream starts to rise. The insulin helps the body to utilize the sugar in its metabolism.

Diabetes

Diabetes is a metabolic disease. The chemistry of the body has become upset. The exact cause is not known and the whole process is not understood. The most striking feature of diabetes is the inability of the body to handle sugar. This comes from a loss of certain type of cells in the pancreas

called the Islets of Langerhans. Normally these cells produce a substance called insulin, coming from the Greek word meaning island. When this substance is not present in the body, the body cannot utilize sugar. Also, when the body cannot utilize sugar, it in turn is unable to burn up fat. For some reason in using fat a certain amount of carbohydrate has to be used at the same time. In treating these cases, the doctor watches the diet to see that they get a proper proportion of sugars and of fats. If necessary he prescribes insulin which the patient gives to himself by hypodermic injection. With modern methods it is generally only necessary to give one injection a day, although some patients still have to take three injections a day.

With modern treatment of diabetes these patients live much longer. However, the control of the blood sugar is not the whole story. Diabetes is a complicated disease and is a degenerative process. As part of this picture these patients generally show a diseased process going on in the blood vessels which is probably the same as arteriosclerosis in any other person, and this carries with it all the effects of arteriosclerosis. These people are more susceptible to infection, and because of this when eye surgery is done on one, care must be taken and they must be watched with extra zeal. They are more prone to infections elsewhere in the body, as carbuncles on the neck or infections on their feet, and they have to have special care in handling their feet. If their vision is poor, it is necessary that someone will inspect their feet nearly every day to see that the toenails are properly cut and are not digging into the adjacent toe, and that no infection is starting.

The eyes of the diabetic person are more prone to a number of condi-

tions. They seem to be more susceptible to iritis. They may get a type of iritis called rubeosis irides, in which the iris becomes highly vascularized. Treatment here is very difficult. The pigment epitheleum on the back of the eyes degenerates in these people so that it is very friable, and when the surgeon opens the eye to do a cataract operation, the pigment breaks free and is washed in the aqueous. When a surgeon sees this he is at once suspicious of diabetes. The diabetic is also more prone to develop cataracts and will frequently develop a cataracta complicata which, as was described, is an opacification that develops mostly in the back of the lens right against the posterior capsule. The eyegrounds of a cataractous patient are very apt to show hemorrhages. These hemorrhages instead of being "flame shaped" such as are seen with a hypertension, are generally little round spots and there will also be a white exudate which is seen as a little white hard-looking spot. These generally occur in groups. The arteries generally show an arteriosclerosis. When the condition progresses more hemorrhages occur and sometimes these hemorrhages push forward into the vitreous. Then, as the hemorrhage in the vitreous is absorbed, new blood vessels grow into the hemorrhagic area. As these blood vessels are very friable, they in turn have fresh hemorrhages. Then again, blood vessels grow forward into these hemorrhages and the process is repeated, so that in looking into the eye the ophthalmologist will see blood vessels growing right up into the vitreous toward him. It looks to him very much like seaweed in the water along the ocean. This is called rete mirable. This condition is very serious for it has a tendency to progress, and to destroy the vision of the patient.

The treatment of diabetes, although it helps to keep the diabetic condition itself under control as far as sugar is concerned seems to have very little effect on these changes which occur in the eye.

Hyperthyroidism

In contrast to diabetes where the glands of internal secretion stop functioning, this condition of hyperthyroidism is characterized by an over-activity on the part of the thyroid gland, probably from an overstimulation by a material secreted in the pituitary glands. The material secreted, thyroxin, stimulates the body metabolism making it burn up its oxygen faster over the whole body, much as opening the damper on a stove makes the fuel in the stove burn faster. This affects the body in many ways. These patients are keyed up. They respond much more rapidly, there is a tremor to their hands and to their tongue. They like cold weather and need very few blankets in the wintertime, and are nervous and jittery. To supply more blood for all of this metabolism the heart has to work much harder. In the eyes, the most prominent symptom is an exophthalmus. The cause of this exophthalmus is complex. Partially it is due to an increase in the tonicity or tension of the muscles, both the regular extra-ocular muscles and the smooth muscles that are in the orbit. Partly the appearance is due to the lids being held wider apart, due again to the increased tonicity of the muscles. It is also caused by an edema of the extra-ocular muscles and of the orbit. This edema is probably caused in some way by a material secreted by the pituitary gland. In a lesser degree this exophthalmus is not harmful, although the patient does not like the expression it gives to his face. As it continues, the lids are unable to completely cover the eye. When this occurs, the cornea is gradually left exposed with the dire results which follow exposure. The tears cannot wash the cornea and moisten it. The cornea drys, grows lusterless, and in time will ulcerate. In hyperthyroidism there are other effects on the eye which are of clinical importance in diagnosing the condition.

These include a lag of the upper lid when the patient rotates his eyes downward from a position of looking up. As he looks down, the eyeball rotates downward faster than the upper lid. This exposes the sclera, or white of the eye above and is called lid lag.

This condition is treated by surgical removal of part of the thyroid gland, sufficient being left to carry on the normal metabolism of the body. In some instances the exophthalmus will continue to progress even after the thyroidectomy. Here some surgical procedure is necessary to relieve the pressure in the orbit so that the lid can be brought over the eye.

Parathyroid Gland

Sometimes in performing a thyroidectomy, the surgeon will inadvertently remove some small glands called parathyroid glands which lie just in back and alongside of the thyroid gland. These glands help the body to control the amount of calcium present in the blood, and when they are inadvertently removed, the blood calcium falls rapidly and the patient, if the condition is unchecked, goes into convulsions. It can be controlled by administering calcium and an extract of the parathyroid glands. When it is uncontrolled a typical form of a cataract forms in the eyes. When seen very early and given prompt treatment, these cataracts can be arrested so that they will not grow any worse. Once developed, the only treatment is an operation to remove the lens.

Pituitary Gland

Another gland of internal secretion which may affect the eyes indirectly is the pituitary gland. This gland is situated behind and below the optic chiasma. Tumors of this gland will present the eye signs common to any

cancer and to cranial tumors, such as papilledema. Also it presents characteristic changes in the visual field as it grows up from below and generally behind causing pressure on the optic chiasma and the tracts behind it. The most characteristic field is the loss of the temporal half of the field of vision in each eye. This will start in the upper temporal portion, then extend to include the lower temporal portion, then come around to the lower nasal portion, and then finally wipe out the whole visual field. A variety of patterns may occur, however, depending upon the exact location of the tumor.

Diseases of the Blood

Anemia is a term used to describe a deficiency in the amount of hemoglobin in the blood cells. This hemoglobin is a material which carries oxygen to the tissues of the body. It also gives the blood its red color. An anemic person has a pale color to his skin and conjunctiva because of this lack of hemoglobin. On examining the fundus of an anemic person, the fundus also has the same pallor. Because of the lack of oxygen, the tissue becomes devitalized and hemorrhages occur into the retina. Also at times white deposits very similar to those seen in arteriosclerosis occur. If the anemia is severe enough, an optic atrophy can occur.

Leukemia is a condition in which the white blood cells of the body start growing rapidly. This is really a form of a cancer. The fundus examination here, shows engorgement of the veins, also white deposits in the retina and hemorrhages. These hemorrhages at times, instead of being just a red color will be red with a white center.

DISEASES OF THE OPTIC NERVE

The optic nerve forms the connection between the eye and the brain. It is really a pathway of the brain rather than a "nerve," since embryologically it began as a connecting stalk between the wall of the brain and the early outpouching of brain structure which later forms the retina of the eye. Because of this difference in structure from other nerves of the body, the optic nerve reacts differently to injury. A so-called "peripheral nerve" of the body which carries either the stimulus for movement of a muscle or a certain type of sensation to the brain from the outside world is able to repair itself after injury. Thus a severed nerve, if treated properly, can grow back together and eventually function again. When the optic nerve is damaged, it has no such powers of regeneration, and the portion which is affected never functions again.

The optic nerve is made up of about 1 million individual nerve fibers most of which originate in the retina, and run toward the point of attachment of the nerve to the back of the eyeball. These are gathered together in bundles to form the optic nerve which then passes backward to the brain. The nerve proper is only about $1\frac{1}{2}$ millimeters in diameter, slightly less than a common kitchen match, but its outer protective coverings make it seem much larger as viewed in the dissection room.

Certain congenital anomalies can be present in the optic nerve. By "congenital" we mean that the condition is present at birth not that it is hereditary. Some congenital abnormalities are indeed inherited, but the use of the word "congenital" does not imply this. A rare defect may occur in which a portion of the optic nerve is absent. This can be discovered by the physician who examines the interior of the eye, and is called a coloboma of the optic nerve head. There will be a defect in the field of vision which

corresponds to the portion of the nerve which is missing. More frequently there may occur medullated nerve fibers which have a whitish coloration as compared with the remaining retina. The normal nerve fibers in the retina have a surrounding coating or sheath which is transparent. After the optic nerve leaves the eyeball, these same nerve fibers are covered with a sheath of whitish material which acts somewhat like insulation around a wire. This substance is known as myelin, and a nerve fiber with such a covering is called medullated. When medullated nerve fibers occur in the retina they are not transparent and the light impulses cannot get through them to reach the deeper-lying rods and cones. This produces a blind area in the field of vision which corresponds to the patch of nerve fibers involved. When such a patch lies over the macula, central vision never functions. If the area of abnormal fibers is located elsewhere, the defect in the field of vision may never be discovered.

Inflammation of the optic nerve is known as optic neuritis. It may occur either where the nerve is gathered together from the retinal nerve fibers, or anywhere along the path of the nerve as it leads back toward the brain. If the inflammation strikes at the point of exit of the nerve from the eye, changes occur which are visible to the examining physician. These consist of swelling of the nerve head, hemorrhages in the nerve and surrounding retina, and a partial blockage of the veins leaving the retina. If the process occurs farther back in the nerve away from the eyeball, the interior of the eye may look perfectly normal. In either case the symptoms are much the same. The patient usually has a fairly sudden loss of vision in the affected eye. This is because the fibers from the center of the retina, the macula, are more susceptible to damage, and their loss of function leads immediately to a loss of central vision. The visual field shows a blind area exactly in the center, corresponding to the loss of function of the macula.

In very severe cases even the outer rim of visual field may be lost. One of the more common causes of optic neuritis in the colder northern climate is a generalized neurological disease called multiple sclerosis. In this condition optic neuritis may occur many years before any of the other symptoms appear. Recovery from the attack of optic neuritis is expected in multiple sclerosis, but attacks may recur, and with each one a certain amount of permanent damage to vision occurs.

Recovery from optic neuritis is not always predictable. Treatment is usually aimed at the possible infection and is designed also to increase the blood supply to the inflamed nerve. There is generally a certain amount of destruction of the optic nerve which is later visible to the examining physician as a paleness of the nerve head inside the eyeball. This paling is known as optic atrophy and it indicates permanent destruction of the nerve fibers which have been involved.

If the inflammation has been located at the point of exit of the optic nerve from the eyeball, the condition is known as intra-ocular optic neuritis, and the resulting atrophy of the nerve is called secondary (or post-neuritic) optic atrophy. This term is used because the physician can see evidence of changes due to inflammation around the optic nerve. If the inflammation has been situated farther back in the nerve the condition is known as retro-bulbar optic neuritis, and the subsequent atrophy is called primary optic atrophy or simple optic atrophy. In this latter case the only change seen by the examiner is a pale change in the optic nerve head. There are no surrounding changes about the exit of the nerve, since there was originally no inflammation there.

Atrophy of the optic nerve can also result from damage which is not due to actual infection or inflammation. Tumor of the brain is a quite frequent cause. In such a patient the growth of a tumor raises the pressure

inside the skull, and this pressure is transmitted to the optic nerve. A swelling of the optic nerve head is produced which is diagnostic of increased intra-cranial pressure. This is called "choked disc" or papilledema. If such a swelling is allowed to continue unchecked, permanent damage to the nerve fibers results, leading to optic atrophy. In other cases of tumor of the brain, there may be no rise in pressure within the skull, but the growth may press upon the pathways of vision connecting the eye to the brain. If this process is far enough forward in the pathways, optic atrophy will become visible within the eye. In either event, damage to vision results.

The causes of optic atrophy are important to know, since Government figures place almost 15% of cases of recipients of aid to the blind in this classification. 30% of the cases of optic atrophy were stated as due to syphilis, and probably the figure should be higher. Prenatal factors, such as infection in the mother, and hereditary defects of the optic nerve accounted for 5%. Infectious diseases other than syphilis were the cause of over 5%. Various non-infectious diseases accounted for almost 7%. Poisons were listed as the cause for only 2%, as were newgrowths (tumors) of various types. In over 40%, the cause was undetermined.

It is impossible to discuss all of the poisons which will damage the optic nerve. One of the recent text-books lists over 80 different materials which will have toxic effects on the eye, and many of these involve the optic nerve. The two substances which most frequently produce such damage are probably wood alcohol and lead. The former is ingested by individuals whose thirst may overcome their judgment. In some cases innocent persons may be involved by contamination of alcoholic beverages. Lead may be the causative agent in industrial exposure, and in many cases poisoning may occur in children who have chewed the paint off their playthings. There has been considerable progress made in the use of non-toxic paints for children's toys, so this cause

of damage to the eye may gradually become less important. The relatively minor importance of poisons as a cause for blindness can be determined from official figures, which list poisonings as the etiology in only 0.5% of the total number of blind cases summarized.

Tobacco amblyopia should be mentioned briefly. In certain individuals there exists a sensitivity to tobacco which results in the loss of central vision in both eyes. The site of damage is in the nerve fibers which lead from the macula, and if the process is allowed to continue, permanent loss of central vision may result. Early treatment which includes complete abstinence from tobacco often results in remarkable improvement. There is a good deal of argument among various authorities as to whether ethyl alcohol, the type used for beverages, can produce a similar toxic amblyopia. Some argue that excess alcohol intake decreases the amount of food eaten, thus leading to a vitamin deficiency which affects the optic nerve. Others feel that impurities in the alcohol are to blame. In either event, there is no conclusive argument against the use of alcohol providing the food intake is adequate and the beverage of good quality.

TUMORS

The word "tumor" means an abnormal growth of cells somewhere in the body to produce a mass of tissue not ordinarily present. A "benign" tumor is one which grows only in its original location and has no tendency to spread to other and more distant parts of the body. A "malignant" tumor is one which will spread to other regions of the body by a process which is known as "metastasis." In such a spread small groups of tumor cells are carried either by way of the blood stream or by the tissue fluid channels (the lymphatics). They come to rest in some other organ of the body and multiply there to form a new tumor. Such a spread may be very extensive with new areas of tumor growth in many parts of the body.

Tumors interfere with the normal functions of the body either by pressing against vital organs or by growing into them to replace normal tissue. Thus a benign tumor can kill as surely as a malignant one if it is in the proper location.

The reason for the abnormal multiplication of cells into such a newgrowth is the basic mystery which research into the cause of cancer is attempting to solve. The term "cancer" is used very loosely to include usually the malignant types of tumors and has little place in accurate medical terminology. If the tumor arises from cells of the skin, of the gastro-intestinal tract, of the genito-urinary tract, or of glandular structures derived embryologically from any of these it is called a carcinoma. When the newgrowth is derived from the supporting tissues of the body such as bone, muscle, or connective tissue it is known as a sarcoma. In certain special cases where a tumor arises from embryologically younger cells, or where the exact type of cell origin is unknown, other terms are used. Hence the name retinoblastoma for a malignant tumor of the eye which grows from primitive retinal cells, the retinoblasts.

Tumors which affect the eye cause damage either by destruction of visual function or by spreading to vital parts of the body to eventually kill the individual. In many cases both effects occur. Less than 1% of the total group listed by the Government as recipients of aid to the blind are the result of tumor growth. This figure is misleading as to the number of cases affected since it lists only the survivors. If the tumor is not treated early or successfully, the individual does not live to receive blind aid. It would follow that the greater number of patients listed in the Government figures are blind as the result of either tumor pressure upon the optic nerves or by effects upon the visual pathways in the brain. Two-thirds of the cases fall into this group. This would include brain tumors of local effect only in which treatment was successful for preservation of life.

The eye can be affected directly by newgrowths of the skin of the lids, of the cornea, the iris and ciliary body, the choroid, the retina, and the optic nerve, as well as tumors growing within the orbit outside the eyeball. Metastatic tumors which reach the eye by way of the blood stream from other sites in the body can likewise destroy vision.

Carcinoma of the skin of the lids is common. It can be treated early either by excision or irradiation with a high degree of success. Neglected cases occur where the newgrowth has extended to the eyeball or into the orbit. The usual method of treatment in such a case is to surgically remove the involved lids and the entire contents of the orbit. This disfiguring operation is called orbital exenteration. Since this condition is more often limited to one eye only, such patients seldom reach the social worker for the blind.

Tumors of the cornea are usually in the carcinoma group. They may be treated early either by excision or irradiation with X-ray or radium. More extensive ones require either removal of the eyeball (enucleation) or, more often, orbital exenteration. It is puzzling to find that 20% of the cases

blind from tumor in the Government figures are stated to be due to newgrowths involving the cornea, since these are usually found in only one eye.

The iris, ciliary body and choroid are affected by a tumor known as malignant melanoma. This is one of the most deadly newgrowths found anywhere in the body. Metastasis to other regions occurs early and often is fatal. When such a tumor occurs on the iris or ciliary body it may be recognized fairly early in its course. In rare instances removal of the section of iris containing the tumor may be successful, but in the majority removal of the eyeball is necessary. Irradiation has little effect on this tumor. When a malignant melanoma occurs in the choroid it may be discovered much later in its course. An associated retinal detachment may mask the tumor, making diagnosis difficult. The tumor growth within the eyeball may cause the intraocular pressure to rise, producing secondary glaucoma. Thus an eye with unexplainable glaucoma is often best removed. Enucleation of the eyeball is again the treatment of choice. Often this is too late since the tumor has already spread to involve the liver or lungs. Few of these cases of malignant melanoma reach the blind rolls since they either involve one eye or lead to fairly early death.

The major tumor of the retina is the retinoblastoma. This is a highly malignant newgrowth which usually affects children before the age of 5 years. Primitive retinal cells, the retinoblasts, proliferate and form a mass in the posterior segment of the eyeball behind the lens. The parents may first discover that the eye does not see or that a peculiar greenish-white glint is visible through the pupil. This is known as the amaurotic cat's eye, amaurotic because the eye is blind, and like the feline eye because of the peculiar light reflection through the pupil. The tumor may produce secondary glaucoma which makes the eye red, hard and painful. The mass of tumor cells may have calcium deposits or even bone formation in its center, so that in some cases

an X-ray film will aid in the diagnosis.

Since retinoblastoma has a tendency to early spread, often enucleation will be too late as a life-saving measure. The successful treatment of one eye may be followed by the appearance of the same disease in the other eye, resulting in total blindness. For this reason a much higher percentage of blindness occurs in young persons as the result of tumor growth. Retinoblastoma is also the leading cause of death among blind children.

There is a distinct hereditary factor in the occurrence of retinoblastoma. Many instances are recorded where several children in the same family are affected. There are a number of cases in which the tumor has been traced through two (and sometimes more) generations. More cases are being seen because early treatment has produced more survivors who can then transmit the disease to their children. There will doubtless be an increasing number of cases of this type on the blind rolls.

In general, removal of the eyeball as early as possible is the best treatment. The second eye should be watched carefully until the age of 9 to 12 years. Survivors should remain childless.

Tumors of the optic nerve, often called gliomas, also are prone to occur in children. Surgical removal results in blindness or absence of the eye involved, and frequently the condition is fatal.

Metastatic tumors of the retina and choroid may be carried to the eye by way of the blood stream from many sites in the body. One of the more common is tumor of the breast. By the time the spread has been sufficient to reach the eye, the patient has a hopeless outlook, so practically none reach the social worker with the blind.

It will be seen, then, that treatment of tumors, either benign or malignant, consists of their early discovery and destruction. This can be done

either by complete surgical removal or by the rays of X-ray or radium. In either case, destruction of vision occurs. The basic question: "What makes tumor cells grow?", is still unanswered.

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